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REDAKSIONEEL · EDITORIAL

'N ENTSTOF TEEN MASELS?

Die enigste biologiese profilakse-tegnieke wat, tot dusver ernstig oorweeg, teen masels toegepas kon word, was die gebruik van herstelserum, die gebruik van gamma-globulien voorberei van saamgevoegde plasma of die globulien-fraksie verkry van vars placenta. Daar word beweer dat die gebruik van beskermende dosisse serum van 'n goedgekeurde sterkte 'n tydelike onvatbaarheid gedurende 'n tydperk van 2 tot 3 weke bewerkstellig het by kinders onder 3 jaar, by swakkes en verswaktes, by pasiënte van enige ouderdom wat aan ernstige akute of chroniese kwale ly, en tydens uitbrekings in inrigtings. Daar word ook gemeen dat die maselsaanval aansienlik gewysig kan word deur die gebruik van onvatbareserum of sy globulienfraksie.

Aktiewe immunisasie teen masels is reeds so lank gelede soos 1758 op die proef gestel toe Home die blood afkomstig van 'n velletsel by 'n pasiënt wat aan masels gely het, aan die oopgeskraapte armvel van 'n kind aangewend het. (Jenner was toe maar 9 jaar oud). In die afgelope jare het ander werkers afskeidings uit die boonste asemhalingskanaal in die neusweë van vatbare pasiënte geplaas. 'n Binnehuidse inenting is ook op die proef gestel. Lewende entstowwe is voorberei van soorte wat verswak is deur serie-oorgang in eiers. Geen algemeen aanneemlike metode vir aktiewe onvatbaarmaking is egter tot dusver deur die mediese professie toegepas nie.

In Maart vandeesjaar, op 'n konferensie van viroloë in die Verenigde State, het Enders (aan wie 'n Nobel-prys toegeken is vir sy weefsel-

A VACCINE AGAINST MEASLES?

Until now the only seriously considered biological techniques of prophylaxis against measles have been the use of convalescent serum, the use of gamma globulin prepared from pooled plasma or the globulin fraction obtained from fresh placenta. In children under 3 years, in the weak and in the debilitated and in those of any age suffering from serious acute or chronic disease and in institutional outbreaks, protective doses of serum of approved potency have been claimed to confer a temporary immunity of about 2 to 3 weeks. It is also thought that the attack of measles might be considerably modified by the use of immune serum or its globulin fraction.

Active immunization against measles was attempted as long ago as 1758 when Home applied blood from a skin lesion in a case of measles to the scarified arm of a child. (Jenner was only 9 years old at the time). In more recent years other workers have introduced upper respiratory tract secretions into the nasal passages of susceptible subjects. An intradermal inoculation has also been attempted. Live vaccines have been prepared from strains attenuated by serial passage in eggs. No generally acceptable method of active immunization has yet, however, been adopted by the medical profession.

In March this year, at a conference of virologists held in the U.S.A., Enders (awarded a Nobel prize for his tissue culture work which laid the foundations for the Salk poliomyelitis vaccine) reported on the growth of the measles virus in tissue culture first using human kidney

kwekingswerk wat die grondslag van die Salk-poliomiëlitisentstof gelê het) verslag gedoen oor die groei van die maselsvirus in weefselkweking waarvoor menslike nier- en amniotiese selle eerste gebruik is, waarna hy ook in staat was om dit in hoendereiers te groei. Sy resultate dui daarop dat 'n verswakte virus geproduseer is wat teenstowwe te voorskyn kon bring by bobbejane sonder om die siekte self te veroorsaak.

Hierdie fundamentele navorsingswerk kan bes moontlik op die ontdekking van 'n beskermende entstof teen masels uitloop. Enders en sy Harvardspan voel egter nog besorgd oor die moontlikheid dat die verswakte virus harsingskoors kan veroorsaak. Wanneer die gevaar van hierdie ernstige komplikasie uitgeskakel is (en die tussentydse resultate oor hierdie aspek van die probleem is bemoedigend) sal 'n enstof teen masels beslis in sig wees.

Die emosionele geesdrif waarmee Enders se mededeling toegejuig is, word soos volg op bladsy 28 van die uitgawe van *Time* vir 10 Maart 1958 gerapporteer:

‚Selfs met behulp van 'n luidsprekerstelsel was die sagte stem van navorsingswerker Enders skaars hoorbaar op verlede week se vergadering. Maar toe hy klaar gepraat het, het Cincinnati se dr. Albert Sabin uitgegil: „John, jy het dit weer reggekry!" Die vergaderde viroloë het uit gelid getree, opgestaan en hom toegejuig.'

and amniotic cells, after which he was able to grow it in hens' eggs. His results indicate that an attenuated virus was produced which could evoke antibodies in monkeys without producing the disease.

These fundamental investigations may provide a protective vaccine against measles. Enders and his Harvard team are still concerned, however, about the possibility that the attenuated strain may produce encephalitis. When the risk of this grave complication has been excluded (and the interim results on this aspect of the problem are encouraging) the vaccine for measles will definitely be in sight.

The emotional enthusiasm which greeted Enders' communication is reported in *Time*, 10 March 1958, page 28, as follows:

'Even with the aid of the public-address system, soft-spoken Researcher Enders was scracely audible at last week's meeting. But when he had finished, Cincinnati's Dr. Albert Sabin yelled: "John, you've done it again!" The assembled virologists broke ranks, stood and cheered him.'

THE DIFFICULT DUODENUM*

A. LEE MCGREGOR, M.CH. (EDIN.), F.R.C.S. (ENG.)†

Honorary Research Associate, Department of Surgery, University of the Witwatersrand

Death following gastrectomy occurs rarely. When it does, it is often due to a leak developing in the stump of the duodenum with the formation of a duodenal fistula, loss of high intestinal content with electrolyte disturbance and auto-digestion of the skin.

The causes for this train of events are:

1. Imperfect technique in closing the duodenum.
2. Failure to suit the site of the distal section in gastrectomy to the pathological condition present.
3. Unsatisfactory drainage of the stomach after operation.

1. TECHNIQUE IN DUODENAL CLOSURE

Proper closure of the duodenal stump depends in the first place on having enough of the duodenal tube mobilized to admit of satisfactory closure and secondly on doing it in the correct fashion. A useful principle in gastrectomy is to work from the healthy towards the diseased area. Thus for gastric ulcer the operation begins at the duodenum which is cut across. In duodenal ulcer the section is made through stomach first. In either case the surgeon is left with a 'handle' of stomach which he can manipulate and which much facilitates the more difficult dissection in the neighbourhood of the pathological area. In duodenal ulcer cases the back of the first part of duodenum is thus more readily exposed and it can be freed from the subjacent pancreas. The posterior surface of the first part of the duodenum is uncovered by peritoneum and the bowel is friable and readily injured. This is one of the most vascular areas of the body and requires the utmost gentleness in exposing and ligating the small vessels entering the bowel. For this purpose mosquito forceps are used and unabsorbable ligature material such as No. 60 linen thread. This is mandatory as an accessory pancreatic duct is sometimes unknowingly divided, or small portions of pancreatic tissue may need tying. If catgut is used it is speedily dissolved by tryptic ferments. It was the realization of this fact which has made operations on the pancreas safer.

It is the surgeon's aim to secure a mobilized portion of duodenum distal to the line of proposed section which admits of safe closure. He

* The third article in the series *Operations of Election*.

† Consulting Surgeon, Johannesburg General Hospital.

strives at a length of half an inch but can manage with less. The last area to be mobilized is the upper border of the duodenum, where the right gastric artery is tied and cut. The duodenum is now divided distal to the pylorus. A clamp such as the Allen type (a Kocher's artery forceps will do) is now put across the bowel in such a fashion that it cannot slip (Fig. 1). A small Payr clamp is applied just proximal and the duodenum divided with a cautery. Both these clamps have a spike at the end and this is made to grip the bowel wall. The duodenum is now ready for closure. A 000 atraumatic chromic catgut suture on a curved (shilling size) round bodied needle is now put in. The suture has been lubricated with sterile paraffin.

It commences at the superior border and is a continuous running mattress suture taking both serous and muscular coats in the bite. The first bite having been taken is tied and a forceps applied to the free end. The suture is made across the clamp. When completed the clamp is disengaged and withdrawn and the suture tightened. This inverts the cut end. The duodenal end is pushed on to the suture traversing it which shortens the cut end of the cone (Figs. 3A, B and C).

This suture is not tied but returns to its starting point also as a mattress suture which further inverts the bowel end. When ap-

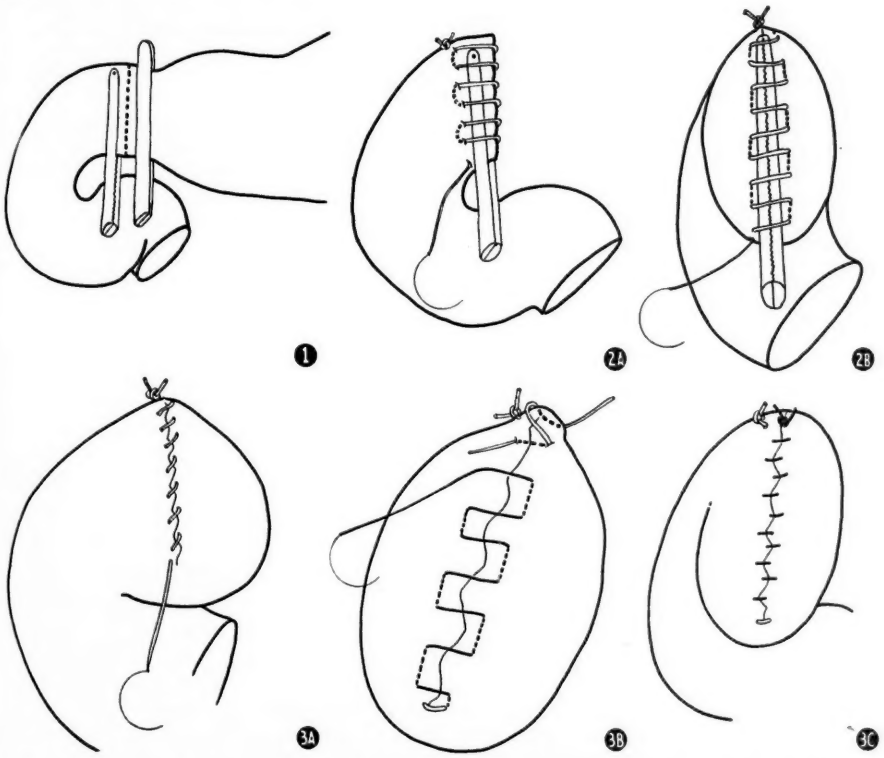


Fig. 1. Dividing the duodenum. The line of section is shown. Both clamps are distal to the pyloric sphincter. The clamp on the reader's right is a small Payr, that on the left is an Allen clamp. Note that this clamp stops short of the upper margin of the bowel and that the spikes at the end grip the duodenum and prevent slipping.
Fig. 2*a*. Closure of the duodenum (side view). The first row of sutures is being applied across the clamp. The needle should be curved.
Fig. 2*b*. Closure of the duodenum. End view of the first line of running mattress suture.
Fig. 3*a*. Closure of the duodenum. The clamp has been removed. The suture has been pulled tight.
Fig. 3*b*. Closure of the duodenum. The second row of the running mattress suture has been put in by returning the first suture towards its starting point. Before completing the stitch, the projecting corner of the bowel is being inverted by a separate Z suture.
Fig. 3*c*. Closure of the duodenum. The second row of sutures is completed.

proaching the top of the divided bowel, the latter will often project in a small cone. The returning suture is temporarily discontinued and a separate Z suture put in to invert the cone (Figs. 3A, B and C).

The second layer of suture is now completed over the inverted cone and tied off. Observe that the commencement of this suture has been buried by the Z suture. This is the reason why this suture is tied off as soon as the first bite has been taken.

A layer of No. 60 linen sutures is now put in as interrupted mattress stitches which still further invert the duodenal stump. It is a rule when dealing with duodenum or pancreas to use unabsorbable suture material for the reason already given.

Errors in the Closure. The security of the stump may be endangered in several ways:

(*a*) By insufficient gentleness which damages and devitalizes tissue in an area bathed in strong digestive juices.

(*b*) By using a clamp which slips off the bowel and makes closure difficult.

(*c*) By using coarse or unsatisfactory suture material.

(*d*) By failing to use an outer layer of unabsorbable sutures.

THE DIFFICULT DUODENUM

The hazards associated with the duodenum in the operation of gastrectomy may be classified as follows:

1. *Noli me tangere*—the duodenal condition forbids direct attack.
2. Difficulties associated with the actual closure of duodenum.

1. PRE-PYLORIC SECTION OF STOMACH

It happens, fortunately infrequently, that there is so much active inflammation at the ulcer site and so much reaction around that the first part of the duodenum presents as a pink indurated mass. It would be not only unwise but dangerous to make a direct attack on this area. The surgeon may choose one of several procedures:

(*a*) Quite the best is to plan operation so that it is not undertaken during the stage when the ulcer is active. It is gratifying to see the change which ensues on a preliminary period of medical treatment and we have, since adopting this plan, not been faced with the need to carry out a restricted operation. Prior to this it has been necessary to do a pre-pyloric section in 7 of 154 operations for ulcer. In these cases where follow up extends on an average of three years the results have been completely satisfactory.

(*b*) When confronted with an inflamed, swollen, indurated duodenum it may be dealt with by a one- or a two-stage procedure.

i. The Finsterer or Bancroft operation is based on the fact that the antral mucosa produces a ferment which is liberated by food impinging on its surface. This ferment is carried by the blood stream to the acid-secreting cells in the fundus and body of the stomach and acts as the trigger for the liberation of acid. Removal of the antral mucosa removes the trigger zone.

Technique: The operation mobilizes the curvatures of the stomach up to the site of proximal section and divides the organ. The great omentum is detached towards the duodenum and the lesser omentum is similarly dealt with until the stomach is freed up to the pylorus. The important decision now is to choose the site of pre-pyloric section. The tendency is to divide stomach too near pylorus. Four finger-breadths of stomach should be left *in situ* proximal to the pyloric sphincter. A clamp is applied proximal to the line of section and the stomach removed. The open end of the pyloric antrum is picked up at four points with Allis forceps.

The mucous membrane is dissected off the muscle coat by blunt dissection, care being taken to clip and tie the strands of tissue passing between mucosa and muscle. These are fine blood vessels and are tied with 000 catgut. The stripping is done with blunt pointed scissors while the surgeon and assistants hold the Allis forceps applied to the muscle coat. Great gentleness is necessary to prevent tearing the muscle. The procedure continues until a cone of mucosa is freed up to the pyloric sphincter. It is then amputated with curved scissors. A common mistake is to tie off the apex of the mucosal cone with a ligature.

This procedure may well lead to rupture of the pyloric stump and formation of a duodenal fistula, because bleeding takes place into the shut off cavity and causes tension in an area with an already decreased blood supply to its thin muscle wall (Figs. 4A, B and C). There is no drainage for effused blood and the bag may burst.

The open end of the pyloric area is closed by three layers of sutures. The first is a Connel stitch of 000 catgut on a straight needle which inverts the muscle bringing serosa to serosa. A separate Z stitch is used to invert the projecting end which forms where the suture ends.

The second layer is also of catgut on a curved needle and is a running Lembert suture which produces further inversion. The third

layer is of No. 60 linen thread which is unabsorbable and consists of a series of interrupted mattress sutures. No endeavour is made to invert the walls of the stomach stump so much that a solid ball of inverted muscle results.

ii. The 2-stage procedure as devised by McKittrick of Boston is reported on as giving good results. We have no personal experience of it.

The first-stage operation is similar to the exclusion one for cancer. The stomach is divided proximally at the junction of upper fourth with distal three fourths. The distal end is inverted and closed by two layers of catgut sutures. To the open proximal end the upper jejunum is anastomosed by the usual retrocolic no loop Polya type of anastomosis using a Hofmeister valve. Six weeks later the abdomen is re-opened and the actual gastrectomy is performed. It is reported that the procedure is not attended with difficulty. Section distal to the pylorus is readily carried out and the duodenum closed in the customary way.

DIFFICULTIES ENCOUNTERED IN CLOSING THE DUODENUM

(*a*) *Tearing the Duodenum.* In spite of gentleness and the exercise of care, it may happen, rarely, that the duodenum is torn. When there is considerable stenosis at the site of ulceration of old standing, the efforts to dissect this off the pancreas may cause a tear in the duodenal wall.

Several possibilities ensue:

i. It is sometimes possible to invert such a conical duodenum by purse-string sutures (Figs. 5A, B and C).

ii. The tear in the duodenum may actually be an advantage, so much so that occasionally the duodenum has actually been cut across, whether a tear exists or not, so that the surgeon may insert his left forefinger into the lumen of the bowel. He then has an important guide to dissection.

The danger in these cases is injury to the horizontal part of the common hepatic artery. If the dissection adheres closely to the duo-

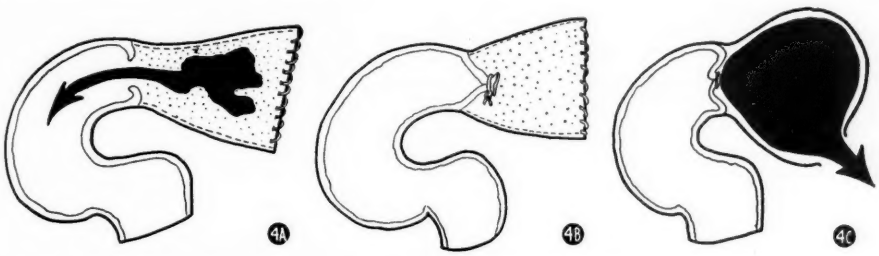


Fig. 4*a*. Pre-pyloric section of the stomach. The correct way to deal with the stump. Note that the seromuscular coat has been sutured. The mucosa has been cored out and cut across. This allows for effused blood to find its way into the bowel.
Fig. 4*b*. Pre-pyloric section of the stomach. Incorrect closure. The core of mucosa has been ligatured, leaving a closed space.
Fig. 4*c*. Pre-pyloric section of the stomach. Incorrect technique. Effused blood has burst the suture line and a duodenal fistula will result.

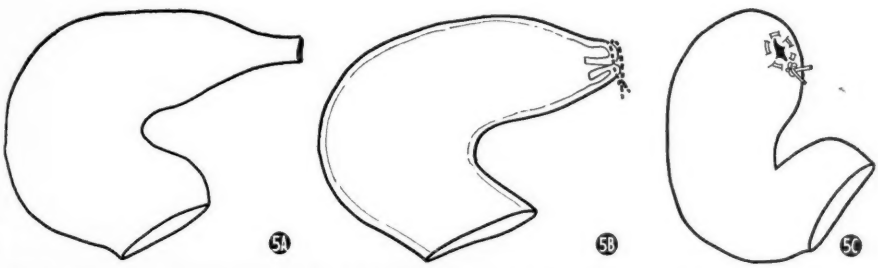


Fig. 5*a*. Closure of an attenuated duodenal stump. The type of stump which admits of purse-stringing.
Fig. 5*b*. Closure by purse string suture. The first purse-string has been inserted and tied over the inverted duodenum.
Fig. 5*c*. The second purse-string has been tied.

denal wall the vessel will not be injured and a finger in the lumen supplies a valuable guide. It is then possible to free enough of the duodenal stump to permit of adequate closure.

(*b*) *Scarring of the Duodenal Wall.* With posterior wall ulceration it is at times inadvisable to endeavour to dissect the viscus from the pancreas as this would result in a gap in the posterior duodenal wall. The following method has been found valuable. The duodenum is cut across through the area of fibrosis or ulceration. The closure is effected by the 'pixie cap' method:

i. The anterior wall is inverted longitudinally for an inch or so (Figs. 6A, B and C).

ii. A series of No. 60 linen sutures on a fine needle is put in thus: the needle picks up anterior duodenal wall lateral to the inverted area and then passes through the dense scar tissue on the posterior wall, thus further inverting duodenum.

We have found this an eminently satisfactory way of dealing with these difficult cases.

(*c*) *Inadequate Duodenal Closure.* It happens, albeit infrequently, that the surgeon finds he cannot make an adequate closure of the stump. The difficulty in these cases is that there is thickening of the posterior duodenal wall and it takes stitches badly. They tear the wall and pull out. It is then unwise to apply more stitches which will just make matters worse. Faced with this exigency there are 2 courses available:

i. If the closure is fair, a Penrose drain is put down behind the duodenum and brought out through a stab wound in the flank. Provided no pressure is allowed to build up in the duodenum, this method is usually adequate. Safety-valve drainage of this type should be used whenever the least doubt exists of the adequacy of the duodenal closure.

ii. If the closure is patently inadequate, a No. 16 Wishart catheter is put through the open end of the duodenum which is then narrowed round the catheter by a purse string or other type of suture(s). A portion of omentum is put round the junctional area and fixed with a stitch or two to neighbouring tissues. A Penrose drain is put into the hepato-renal pouch. It and the catheter are brought out through a stab wound in the flank.

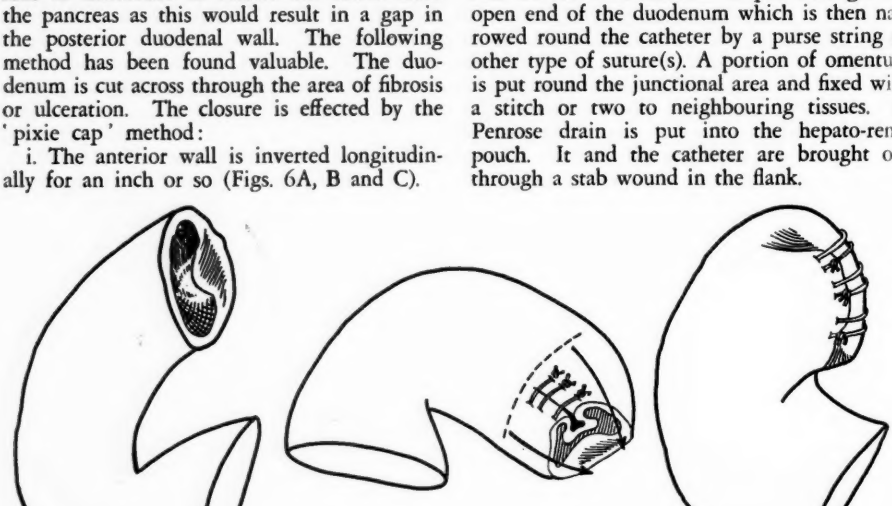


Fig. 6*a*. The duodenum has been divided through the base of the ulcer.
Fig. 6*b*. A portion of the anterior duodenal wall has been inverted by a series of sutures placed transversely. The duodenum will be further inverted by sutures, bringing the dotted line to the base of the ulcer as shown by the arrows.
Fig. 6*c*. The closure complete.

On the 3 occasions when we have used this method, the leakage has been slight, the convalescence no longer than usual and the outcome entirely satisfactory.

We feel emphatically that such cases do well because no back pressure is allowed to build up in stomach or duodenum because ideal drainage is secured by making the Allen type of jejunostomy-stomach drainage at the end of the operation (a catheter is sewn in the upper jejunum. One end passes through the gastro-enterostomy opening into stomach, the other end comes through a stab wound in the abdominal wall).

THE SITE AND STATE OF THE ULCER IN RELATION TO THE OPERATION

Experience has taught that if the distal line of section in gastrectomy for duodenal ulcer can be made beyond the pylorus the outcome will

be satisfactory in the vast majority of cases no matter where the ulcer lies in relation to the line of section. In other words, it is not essential nor is it feasible to remove the ulcer in all cases. Furthermore, to endeavour to do this will bring added risks to the operation. Thus the need has not arisen in this series of cases to put a T-tube in the bile duct as a pilot. Certainly no further trouble has arisen in the duodenum in these cases.

There have been a number of post-bulbar ulcers in the series. Their removal was impossible and they have given no trouble after operation nor has post-operative or later haemorrhage arisen from this source.

In 3 cases where a fistula existed between bile duct and duodenal ulcer as shown by ingested barium outlining the bile ducts, gastrectomy has been possible with section beyond the pylorus. No endeavour was made to disturb the fistula. These cases were all done several years ago and have had no further trouble ascribable to fistula or ulcer.

Our experience shows that gastrectomy will cure the patient of such symptoms as were occasioned by his original ulcer, whether or not the ulcer itself is removed. The continuous alkalinization of the ulcer-bearing area following gastrectomy is adequate to cure the ulcer which brought the case to surgery.

OPSOMMING

Die skrywer dra 'n verdere artikel by tot sy reeks oor Verkieslike Operasies, en bespreek die chirurgiese probleme wat deur 'n moeilike twaalfvingerige derm opgelewer word.

I thank Dr. E. A. Thomas very sincerely for the figures which illustrate this paper. Their simplicity and clarity are characteristic of his work.

PARALYTIC VIVISECTION

ERIC LURIE, M.B., B.CH.*
Johannesburg

Although tremendous advances have been made in all branches of medicine, the progress in anaesthetics has been phenomenal.

The good name which the specialty of anaesthetics has built up over the years, is now, unfortunately, being jeopardized by a certain fairly new technique which can aptly be termed 'paralytic vivisection,' in which the patient being operated upon is conscious or semi-conscious; feels the endotracheal tube in his throat; feels pain of varying degree from the surgical stimuli; can hear and remember what is said at the operating table. He is awake but completely paralysed with large doses of relaxant drugs and therefore cannot do anything to help himself. It is a gruesome experience for the patient.

Numerous cases of this nature have occurred in this country in nursing homes and hospitals. Many surgeons can vouch for their authenticity.

The patients usually complain to the nursing staff, seldom to the doctor, being too timid to do so. Some even refrain from commenting because they think that perhaps this is the normal course of events.

All these cases of paralytic vivisection have been brought about by the same anaesthetic technique. The patient is induced in the usual way with thiopentone and a relaxant and then intubated. He is next connected to a machine delivering gas and oxygen in varying percentages, usually 25% to 50% oxygen. (Gas and oxygen alone in such ratios, cannot be regarded as an adequate surgical anaesthetic). Controlled breathing is now maintained throughout the anaesthetic. Should the patient gag, move or commence spontaneous respiration, he is immediately given another paralysing dose of relaxant. At the conclusion of the operation the anaesthetist starts to neutralize the paralytic effect of the heavy doses of relaxants (other than Scoline) with large doses of prostigmine and atropine, which are also known to have inherent dangers.[1-3] The patient is awake because he has never really been adequately anaesthetized during the whole procedure.

With this anaesthetic technique the possibility of cases of this nature cannot be denied.

How can one tell whether a patient is awake or conscious when using paralytic vivisection? It is impossible. If the paralysing effect of the relaxant drug were allowed to wear off, the patient would gag, move, react to stimuli and probably walk off the operating table. This is sufficient proof that the patient is inadequately anaesthetized. The relaxant is merely a paralysing agent and has no anaesthetic properties of its own.

* Specialist Anaesthetist.

Furthermore, the very fact that some of the exponents of this paralytic vivisection technique are now seen to be adding various other drugs to their so-called gas and oxygen anaesthetic, such as Trilene, ether, pethidine, additional Pentothal or Fluothane, proves that the method was not satisfactory.

Cases returned to the ward after this technique are often still markedly under the influence of the relaxant drugs. The result is a distressing choking feeling and inability to cough or breathe adequately. Besides being dangerous for the patient, it causes the nursing staff great anxiety.

Nitrous oxide is a very weak anaesthetic agent and we must not expect it to do what it is not capable of doing. There is really no such thing as a pure gas and oxygen anaesthetic; only anoxia and asphyxia, and the higher above sea level the more true this is.

It has been suggested that by paralysing patients with relaxants, the necessary controlled respiration allows the gas and oxygen to be pumped into the patient, thereby full use of the gases being obtained; but surely, if a patient is well anaesthetized and breathing spontaneously, he will also get the gases into his lungs and circulation through his own inhalations. It is not the gases which produce this result but the hyperventilation resulting from controlled respiration. Vigorous hyperventilation will itself produce apnoea and unconsciousness.

There is some evidence now coming to light that prolonged controlled respiration is not so remarkably safe as it was thought to be, because it interferes with the venous return to the heart, with all the attendant consequences.[4]

I do not wish it to be thought that I advocate deep anaesthesia. With modern techniques it is neither necessary nor desirable; but I do feel that every patient should be *adequately* anaesthetized and that relaxants should be used to produce relaxation when the nature of the operation requires it, and not to prevent the patient from getting off the operating table.

I would like to suggest that an adequately anaesthetized patient:

1. Will be unconscious;
2. Will breathe spontaneously when the relaxant drug has worn off;
3. Will not gag, move or react to surgical stimuli when the effect of relaxants has worn off;
4. Will tolerate the endotracheal tube after the effect of the relaxant drug has worn off;
5. Will not feel, hear or remember after induction;
6. Will be fully oxygenated and free of anoxia.

These criteria cannot be satisfied by gas and oxygen alone.

With modern anaesthesia, the patient who has been adequately and wisely anaesthetized, can be brought firstly, to the stage of recovery of protective reflexes and, secondly, even to full consciousness at the conclusion of the operation. The former is essential to the safety of the patient before he leaves the operating theatre and the latter a nice refinement but not vitally necessary. Unfortunately, the competitive spirit makes anaesthetists vie with one another to see who can get their patient awake the soonest and the nearer the awakening occurs in relation to the last one or two stitches, the better the anaesthetist. While there may be something in favour of the skill of the anaesthetist who can do this, one cannot include the paralytic vivisectionists in this competition because their patients have been awake all the time.

The good name of anaesthetics is being spoilt by this paralytic vivisection technique and it is time a halt was called. We should steer a middle course; adequate light anaesthesia, with relaxants added only when indicated.

SUMMARY

Paralytic vivisection has been discussed and condemned.

It is a technique that results in a patient's remaining awake during an operation, but rendered helpless because he is paralysed by relaxant drugs.

A plea is made to halt this practice and to administer adequate light anaesthesia to all patients, using relaxants only when indicated, and not for the purpose of securing patients to the operating table.

OPSOMMING

Paralitiese viviseksie word bespreek en veroordeel.

Dit is 'n tegniek wat tot gevolg het dat 'n pasiënt tydens 'n operasie by sy bewussyn bly. Hy is egter hulpeloos, want hy word deur ontspanningsmiddels verlam.

'n Pleidooi word gelewer dat daar van hierdie praktyk afgesien moet word en dat alle pasiënte met 'n doeltreffende, ligte verdowingsmiddel behandel moet word. Ontspanningsmiddels moet alleen toegedien word as daar indikasies vir die gebruik van hierdie middels bestaan. Hulle moet nie toegedien word bloot om die pasiënt hulpeloos op die operasietafel te hou nie.

REFERENCES

1. Burchell, G. B. (1957): Brit. J. Anaesth., **29**, 127.
2. Hunter, A. R. (1956): Brit. Med., J., **2**, 572.
3. Jowell, D. M. and Wood-Smith, F. G. (1957): Brit. J. Anaesth., **29**, 133.
4. Pope, E. S. (1957): Anaesthesia, **12**, 405.

ACUTE POLIOMYELITIS*

A STUDY OF THE CLINICAL MANIFESTATIONS OF FIFTY CASES SEEN AT THE CHILDREN'S HOSPITAL, JOHANNESBURG, DURING THE 1948 EPIDEMIC WITH SPECIAL REFERENCE TO THE MANAGEMENT IN THE ACUTE PHASE

J. L. BRAUDO, M.B., B.CH., M.R.C.P. (EDIN.)
Johannesburg

(*Continued from p. 498*)

The Effect of Physical Activity on the Course of Poliomyelitis. Russell,[79, 80] using a clinical and statistical approach to this problem, collected detailed data on 100 patients, chiefly adults. He found a remarkable and highly significant increase of severe paralyisis in association with a continuance of physical activity in the prodromal and pre-paralytic 12 hours of the disease. Furthermore, the paralysis was worse in those muscles which were most exercised at the onset of the illness. Horstmann[81] studied 411 patients with poliomyelitis and found that the critical time, as far as physical activity was concerned, seemed to be the first 24–48 hours of the invasive phase of the disease. Very few patients gave a history of severe exertion on the day before the onset of the disease. The fact that there was no correlation between degrees of physical activity and severity of paralysis among the patients with bulbar poliomyelitis raises problems with regard to the meaning of the positive correlation in the spinal cases. She noted a significant increase in the incidence and severity of subsequent paralysis in cases where physical activity was performed after the invasive or second phase of the illness. Correspondingly, a higher percentage of non-paralytic than paralytic patients gave a history of bed rest or minimal activity during the early stages of the major or second phase of the illness.

Albrecht and Locke[82] studied 381 cases in 1949 in Nassau county, New York. They found no correlation between the severity of the paralysis and the activity in the 3 days preceding the onset of the illness—the invasive stage. Age was a major factor in affecting prognosis, since adults irrespective of activity were more severely affected than children.

In this series only 2 cases indulged in strenuous sport on the same day as or the day after the pre-paralytic symptoms began. Case 2 of the non-paralytic variety developed a stiff neck and pain in the right shoulder following a boxing lesson, but paralysis did not occur. Case 8 went swimming in the morning, after having felt sick the day before. Soon after the swim he felt sick again, complained of headache and pain in the neck and, on awakening the next morning, the left side of his mouth was twisted and the food regurgitated through his nose. None of the spinal paralytic group could be attributed to excessive exertion during either the prodromal or invasive phase of the disease.

Since Russell[79, 80] stressed the importance of even normal amounts of exertion—the average activity of a child during the course of the day—in predisposing to paralytic poliomyelitis, a comparison will be made between the number of days ambulant after the earliest prodromata date in the non-paralytic and paralytic group. In 16 cases of the non-paralytic group the onset was sudden and one case was ambulant for one day. The spinal, bulbar and bulbo-spinal group showed the following findings: 19 cases with a sudden onset of paralysis; 2 cases developed paralysis after being ambulant for one day; 6 cases after being ambulant for 2 days; one case after being ambulant for 3 days; 3 cases after being ambulant for 4 days and 2 after being ambulant for 5 days. In the South Australian epidemic Southcott and Crosby[18] analysed 35 paralytic cases in the same way and found that 9 cases started off suddenly, 12 cases after one day; 5 after 2 days; 2 after 3 days; 2 after 4 days and one each on the fifth to ninth days inclusive (Table 12).

The non-paralytic and spinal-paralytic group showed a definite difference in the number of cases ambulant for a period of more than one day. The figures were similar to those

* The References will be published at the end of the concluding article in this series.

of the South African epidemic. Whether the physical activity predisposed to the paralytic form of the disease or whether the patient would have been paralysed in any case is still a problematical question, but the figures, although small, favour the influence of activity.

TABLE 12: DAYS AMBULANT BEFORE CENTRAL NERVOUS SYSTEM INVASION

Type	*Days Ambulant Before Central Nervous System Invasion*								
	0	1	2	3	4	5	6	7	8	9
Non-Paralytic (17 Cases)	16	1	–	–	–	–	–	–	–	–
Paralytic (Spinal, Bulbo-spinal and Bulbar) (33 Cases)	19	2	6	1	3	2	–	–	–	–
Paralytic South Australia Epidemic (Southcott & Crosby, 1949) ..	9	12	5	2	2	1	1	1	1	1

The practical importance of these figures lies in the handling of minor ailments during a poliomyelitis epidemic. All persons with fever of unknown cause, sore throat, gastro-intestinal disturbances, etc. should be kept in bed, or very quiet, for at least 6–8 days during an epidemic in order to minimize the factor of physical activity in the production of the paralytic variety of the disease.

Long Journeys. All the children in this series came from Johannesburg and the distance between their homes and the hospital fell within the 1–7 mile range. In the 1944 and 1947–8 South African epidemics many cases were transported to Johannesburg by ambulance, car or truck over good and bad roads from outlying farms, villages or towns and in some instances the journey sometimes covered 100–300 miles. Despite the fact that only the more extensively paralysed patients were moved, the chances of increasing the severity of the paralysis to include intercostal muscles and the diaphragm, seemed very real in these cases. Their mortality and morbidity rates were higher than those of comparable cases from Johannesburg.[3]

Brahdy and Katz[83] compared the fatality rate in 380 local cases and 113 transported cases. They found the case fatality rate of 5% in the local group and 16% in the transported groups. All deaths occurred in the bulbar or severe encephalitic varieties of the disease and a comparison of the death rates in the 2 groups, only taking into account the bulbar and severe encephalitic cases, revealed the following results: 19 of 78 cases (24%) died in the local group and 17 of 37 cases (46%) died in the transported group. The difference of 22% was statistically significant. Furthermore, 4 local cases died during the first 24 hours after admission as did 9 of the transported cases. This difference was also statistically significant.

There is ample evidence from these data that transportation of poliomyelitis victims from outlying stations definitely increased the mortality and morbidity rates of the disease. The therapy of poliomyelitis should therefore be decentralized in order to remove one of the predisposing paralytic and lethal factors.

How can one explain the relationship between exertion and paralysis? The physical activity may in itself exert a deleterious effect either directly or indirectly upon the course of the disease. If the effect is direct, one must assume that the anterior horn cell can be influenced by its peripheral connexions. There is evidence to support this. Hyden[84] has shown that in the normal experimental animal intense muscular work depletes the anterior horn cells of their protein content. This protein depletion would render the anterior horn cell more vulnerable to attack by the virus. Howe and Bodian[17] found that the susceptibility of anterior horn cells to poliomyelitis virus was altered by section of the peripheral nerve originating in those cells; during the period of neurone regeneration the cell was not susceptible to experimental infection.

There is both clinical and experimental evidence that the virus is present in the body some time before actual paralysis occurs, viz. in the pre-paralytic phase.[26, 27] What determines whether the pathological process in an affected cell stops at a reversible stage or goes on to destruction? Many factors are probably involved: age, degree of immunity of the host or other constitutional factors, fatigue, trauma due to inoculation, operation, injections, violence, strain of the virus and other unknown factors. In some cases the disease is widely destructive from the start whereas in others the pattern is mild and it is conceivable that in the latter case any factor which alters nerve cell metabolism may conceivably lead to further multiplication of the virus and increased cell destruction.

iii. WILL THE PARALYSIS OR THE DISEASE SPREAD?

In this series paralysis spread to involve bulbar or spinal muscles or both in 8 cases. Seven of the cases belonged to the bulbar or bulbo-spinal group and only one to the spinal paralytic. The common clinical features associated with extension of the already existent paralysis were an increase in temperature and pulse rate in 6 cases; an increase in blood pressure in 5 cases of the bulbar variety; an increase in the degree of apprehension and tremor of limbs in 2 cases; a rapid irregular or regular contraction of the alae nasi in 2 cases. Irregularity of the heart beat occurred in 2 fatal cases. Involvement of the respiratory centre at the onset or later in the disease was regarded as an extension of the disease. Two cases commenced with respiratory centre involvement—the one went on to a bulbar palsy and the other developed quadriplegia and palsy of the intercostal muscles and diaphragm.

All the fatal cases showed this extension of the disease. Three demonstrated the rise in temperature, pulse rate and blood pressure, while in 2 a marked increase in the apprehension and tremor were noted. In one case the spread occurred during the night and detailed observations were not available. The disease spread within the first 12 hours in 5 instances, the first 24 hours in 3 instances and the second, fourth and fifth day in one instance each. The disease therefore extended between the period of a few hours and 5 days.

Draper[19] stated that in most cases the initial lesion in the cord was the final one and paralysis rarely advanced. After the eighth day of the illness paralysis was most unlikely. He found that if no additional weakness appeared within 6–12 hours of the first paralysis, there was little chance of any further involvement, but some cases became paralysed as late as the eighth day of the illness. He also drew attention to the graver prognosis associated with the appearance in the patient of increased apprehension and alertness. The significance of the rise in blood pressure in the bulbar group of patients was stressed by Platou.[40] He found that a rise in blood pressure was one of the most valuable signs of the early onset of bulbar involvement. However, more significant was the premonitory marked fluctuation in the blood pressure of patients who were to have bulbar involvement. Blood pressure readings were not recorded frequently enough in this series to confirm this statement.

The association of increased paralysis and a secondary rise in temperature and pulse rate has not been stressed before and occurred in 6 of the 8 cases. This increase occurred as late as the fifth day in this series, but may also be found up to the eighth day of the disease.

1. The important features heralding the extension of an existing paralysis were therefore:

(*a*) A secondary rise in pulse rate, temperature and blood pressure.
(*b*) Increased apprehension, alertness and tremor.
(*c*) Rapid irregular movement of the alae nasi.

iv. WILL THE PARALYSIS BE PERMANENT?

There were 33 cases of paralysis in this series. All cases were re-examined by the author 2 years after the acute illness. Four of the patients died; the results of the remaining 29 cases were analysed. They fell into 2 groups:

(*a*) The bulbar and bulbo-spinal group (10 cases).
(*b*) The spinal paralytic group (19 cases).

The system used to record the state of each muscle or muscle group was that recommended by the Medical Research Council War Memorandum No. 7.[38]

TABLE 13: COMPARISON OF THE SEVERITY OF THE PARALYSIS IN THE ACUTE PHASE OF THE DISEASE AND TWO YEARS LATER

Type	*Severity at Onset*			*Severity 2 Years Later*			
	Mild	*Moderate*	*Severe*	*Normal*	*Mild*	*Moderate*	*Severe*
Bulbar	0	0	7	1	4	1	1
Bulbo-spinal	0	0	3	2	–	–	1
Spinal	0	5	14	8	2	2	7
Total	0	5	24	11	6	3	9

The paralysis was classified in 3 grades:

Severe, in which there was little or no movement at all, corresponding to grades 0, 1 and 2;

Moderate, in which there was a fair amount of movement but insufficient to overcome the effects of gravity, grade 3;

Mild, in which movement against resistance was limited and muscles only tired after excessive use, grade 4. A detailed analysis of paralysed muscle groups is summarized in Table 5.

In the bulbar and bulbo-spinal groups all 10 cases were severely paralysed in the acute stage. Of the 7 cases with involvement of the Xth cranial nerve, all returned to normal but took as long as 6 months to do so in one case. The prognosis of the 7 cases with VIIth cranial nerve involvement differed in that one case remained severely paralysed, one moderately paralysed and 4 cases mildly paralysed, whereas only one returned to normal. The one case with complete involvement of all muscles supplied by the IIIrd cranial nerve recovered completely.

Of the 3 cases with spinal involvement as well, one remained severely paralysed and 2 returned to normal. The final result in the above group was 3 cases with perfectly normal function, 4 mildly paralysed, 1 moderate and 2 severely paralysed. The spinal paralytic group showed the following grades of paralysis in the acute stages, 14 severe and 5 moderate. Follow-up showed that all those in the moderate group returned to normal, whereas only 3 of the severely paralysed achieved normality; 2 remained mildly paretic, 2 moderately so and 7 severely paralysed. The outlook for severely paralysed cases of the spinal variety was worse than that for cases of the bulbar group.

It had long been assumed that the chances for complete recovery of cranial nerve function was almost 100%. In this series 50% of the cases remained with weakness of the facial musculature ranging from severe to moderate in one case each, to slight in 3. The data of 3 reports[86-88] on the prognosis of the cranial nerve lesion together with those in this series are summarized in Table 14.

Sixty-seven cranial nerve palsies (49%) of the 138 palsies returned to normal; 59 (43%) were much improved but showed weakness after exertion of the muscles involved, e.g. crying or laughing in the case of the facial musculature and nasal speech in the case of the palatal musculature; 12 cases or 8% showed severe involvement. The prognosis in cranial nerve palsy was therefore not in keeping with the previous assumption. Eight per cent. remained with permanent severe paralysis but 92% returned to normal for practical purposes.

A critical follow-up study of 50 bulbar cases in the Minneapolis General Hospital showed that between 15 and 17 months after the onset of paralysis of the facial, pharyngeal or laryngeal musculature, significant degree of impairment of motor function were present in 18–77% of cases, depending upon which nerve was affected.[85]

Of the 150 peripheral muscle groups categorized as 10 3 weeks after the onset of the disease, 42 (28%) had improved sufficiently after a period of 2 years to be included in categories 4 and 5, i.e. near normal and normal function. Thirty (20%) of the initial total remained *in statu quo,* whereas 54 (36%) moved into the 1 and 2 brackets and 24 (16%) reached the fair stage. Thus 84

TABLE 14: PROGNOSIS OF CRANIAL NERVE PALSY IN THE ACUTE FORM

No. of Cases	*Cranial Nerve Paralysed*						*Result*																	
							Normal						*Much Improved*						*Slight or No Improvement*					
	III	V	VI	VII	X	XII	III	V	VI	VII	X	XII	III	V	VI	VII	X	XII	III	V	VI	VII	X	XII
																(Grade 4)								
33 Schuhmacher[86]	–	–	–	33	–	–	–	–	–	18	–	–	–	–	–	14	–	–	–	–	–	1	–	–
59 with 67 palsies	3	1	5	37	19	2	2	1	4	16	3	2	–	–	–	20	11	–	1	–	1	1	5	–
Zellweger and Gabathuler[87] ..																								
23 Rascoff[88] ..	–	–	–	23	–	–	–	–	–	12	–	–	–	–	–	10	–	–	–	–	–	1	–	–
10 with 15 palsies	1	–	–	7	7	–	1	–	–	1	7	–	–	–	–	4	–	–	–	–	–	2	–	–
This series ..	4	1	5	100	26	2	3	1	4	47	10	2	–	–	–	48	11	–	1	–	1	5	5	–
Total	(138)						(67)						(59)						(12)					

(56%) of the muscle groups were markedly deficient in power after a 2-year follow-up period (Table 14). Of the 24 groups in grade 1 only 4 (17%) remained in the seriously involved grades, i.e. 1 and 2, whereas 10 (41%) reached the third grade and the same number were categorized as grades 4 and 5. Of the 53 groups graded 2 initially, only 3 (6%) remained *in statu quo,* whereas 13 (23%) moved up one grade and 37 (70%) reached grades 4 and 5. In the original grade 3 group, 1 (3%) of the muscle groups remained unchanged whereas 26 (97%) advanced to grades 4 and 5. In the original grade 4 group, however, the vast majority, 22 (93%) of 24 remained the same, while only 2 (6%) returned to absolutely normal function. In this series one concluded that those muscles severely paralysed (grades 0 and 1) were more likely to remain seriously affected than those in the moderately severe and moderate grades 2 and 3, since only 28% and 31% of the former groups returned to good and normal function as compared with 70% and 97% of the latter group. It was interesting to record the very small number of slightly weakened muscles returning to normal function after a 2-year lapse.

The results in this series were similar to the results in Green's cases as regards those initially regarded as fair, viz. grades 2 and 3. However, in the more seriously involved muscles a much higher percentage of those in this series, viz. 16%, 16% and 12% reached fair, good or normal, as compared with 3%, 0.7% and 0% respectively in the 0 group reported by Green.[89] The reasons for these marked differences are not clear but one factor stands out—the intensity and duration of muscle dysfunction in this series was probably more extensive and consequently accounted for the more severe muscle weakness at the 3-week examination. The examination of muscle groups at that time was therefore more markedly affected by the presence of muscle dysfunction, which probably exaggerated the actual decrease in muscle power.

The eventual outcome of the paralytic cases as a whole has been well documented. Wickman[4] analysed 530 cases 12–18 months after the acute attack and reported 231 cases (44%) as cured and 299 (56%) as paralysed. Lovett and Lucas[90] analysed 213 cases and found 160 (75%) moderately or severely paralysed and 53 cases (25%) mildly paralysed. Jönsson[91] reported 250 cases of which 117 cases (47%) made complete recoveries, and 133 (53%) remained paralysed—the paralysis being mild in 58 cases and severe in 75.

In the 1944–5 Johannesburg epidemic of 150 cases reported there were 102 (68%) complete recoveries and 48 (32%) paralysed cases; the remaining 22 cases died. Of 597 cases analysed in the 1947–8 Johannesburg epidemic 3–6 weeks after the acute stage, 363 cases (61%) had made complete recoveries and 234 cases (39%) had residual paralysis.[5] It was possible that some of the latter cases returned to normal later, but no figures were available.

An analysis of the foregoing figures shows that the eventual outcome of the disease has improved since the early reports of the 20th century, especially those from America. The figures from Sweden in 1905 and 1937 were very similar, as were those in the Johannesburg series of 1944 and 1948 (Table 15).

TABLE 15: MUSCLE POWER ASSESSED 3 WEEKS AND 2 YEARS AFTER THE ONSET OF ACUTE POLIOMYELITIS

Muscle Power After 3 Weeks		*Muscle Power After 2 Years*					
		0	1	2	3	4	5
0	150	30 20%	24 16%	30 20%	24 16%	24 16%	18 12%
1	24	— —	2 8%	2 8%	10 41%	6 25%	4 16%
2	53	— —	— —	3 6%	13 23%	31 58%	6 11%
3	27	— —	— —	— —	1 3%	15 56%	11 41%
4	24	— —	— —	— —	— —	22 93%	2 6%

Very few epidemics followed up over a period of 2 years have been recorded. Apart from Lovett and Lucas,[90] 2 epidemics in Sweden[91, 92] were the only records available.

The percentage of severely paralysed cases in the 3 epidemics was not exactly similar —11% in the Swedish epidemic and 18% in this series; but the figures in Jönsson's series more closely approximated those in this group.[91] The patients ultimately requiring orthopaedic surgery belonged to the moderately and severely paralysed group and therefore ranged from 22–29% of cases in 2 of the epidemics reported in the past 15 years.

Green[89] recorded the improvement in muscles rather than cases over a period of 18 months.

The chances of 'zero' muscles reaching fair, good or normal power were very small (3%, 0.7% and 0% respectively); those of 'trace' muscles increased to 18%, 12% and 2% respectively; those of 'poor' muscles to 23%, 35% and 31% respectively; whereas only 3% of the 'fair' muscles remained stationary, 20% becoming good and 77% normal.

The recovery of muscle power was marked during the first 10 months and particularly during the first 4 months after the onset of the disease. For practical purposes little change was seen after 16 months. This was the result in cases which had been given good treatment.[89] All workers agree that the chances of recovery are best in those cases well treated and supervised in the convalescent stage of the disease.

From the foregoing data it might be concluded that, although occasionally spectacular improvement of severely paralysed cases was seen in all epidemics, the chances of fair recovery of severely weakened muscles were small, and still smaller when good or normal recovery was considered. Fortunately the number of severely paralysed cases was small in this series and the 1948 Johannesburg epidemic. Furthermore, with proper rehabilitation and vocational guidance, most of the children and adults should be able to be economically independent of their relatives and the state.

POST-ENCEPHALITIC AFTERMATH

Post-encephalitic symptoms were present in 8 cases of which 2 originally belonged to the non-paralytic group, 5 to the bulbar and bulbo-spinal group and one to the spinal paralytic.

The mothers complained that the children were very nervous, irritable, cried easily and were difficult to handle (Table 29). These symptoms persisted for from 0.5–2 years. Three cases showed a regression in their bladder and bowel control and took 10 days, 2 months and 6 months respectively, to return to normal. One child became dull and apathetic for 10 days in the convalescent stage only.

Apart from these minor yet very troublesome disturbances, a major psychiatric state occurred in Case 8. Eight months after leaving hospital he tried to commit suicide by hanging himself from the top of the wardrobe in his room. His parents said that this school-going child had great difficulty in re-adjusting himself to his complete facial nerve paralysis, since he was continuously being called 'crooked face' at school. This was further aggravated by his difficulty in swallowing, which persisted for 6 months after discharge from hospital. Furthermore, his sporting activities were restricted and it took 2 years for complete re-adjustment to take place.

The mental adjustment in the other 21 cases was surprisingly good, despite extensive physical involvement. Of the 2 cases with the most extensive paralysis, one became irritable for 2 months after the acute stage had passed, while the other[15] developed none of the post-encephalitic manifestations.

Post-encephalitic phenomena occurred in 7 (16%) of 50 cases. Only 2 of 17 cases (12%) of the non-paralytic variety displayed these symptoms; whereas 5 of 14 cases (36%) of the bulbar group did. The spinal paralytic group had only 1 of 19 cases (5%) with these symptoms.

Very few references to the post-encephalitic phenomena could be found in the literature. Although the encephalitic manifestations, e.g. apprehension, tremor, hyperexcitability, etc. were noted in the acute stage by Peabody *et al.*[30] and Baker,[51] little reference to these symptoms was found in the discussion on prognosis. The post-encephalitic phenomena were most prominent in the bulbar group.

TABLE 16: PROGNOSIS OF PARALYSIS IN THREE SEPARATE EPIDEMICS

	No. of Cases	*Complete Recovery*	*Paralysis*		
			Mild	*Moderate*	*Severe*
Nettleblad[112]	145	22 (16·8%)	97 (67·8%)		16 (11·1%)
Jönsson[75]	250	117 (46·8%)	58 (23%)	47 (18%)	28 (11%)
This series	46	28 (61%)	6 (13%)	3 (7%)	9 (19%)

The investigation of the psychiatric problem of poliomyelitis is still in its infancy but most modern workers are fully conscious of its importance.

SUMMARY

1. The mortality rate in poliomyelitis varies from 4–12% but occasionally reaches the high figure of 37%. In this series 4 patients (8%) died. The incidence of bulbar poliomyelitis was higher in the male sex.

2. The diphasic clinical pattern had a more serious prognosis than did the other two patterns.

3. Marked encephalitic symptoms, hypertension and pronounced tachycardia in the absence of hypoxia indicated a grave outcome.

4. There was no way of anticipating paralysis.

5. The influence of physical activity, inoculations and injections, tonsillectomy and long journeys are discussed in detail.

i. The continuance of physical activity in the prodromal and pre-paralytic 12 hours of the disease predisposes to a highly significant increase of severe paralysis in adults. In this series (all children) the deleterious effect of physical activity in the prodromal phase was also confirmed.

ii. The etiological role of intramuscular injections in paralytic poliomyelitis has not been satisfactorily established. Most authorities favour withholding anti-diphtheria inoculations in epidemics—in some communities diphtheria epidemics have subsequently taken place. In this series the causal relationship was not striking.

iii. Tonsillectomy should never be performed during epidemic poliomyelitis.

iv. Long journeys are deleterious in severe poliomyelitis.

6. Certain features heralded extension of the disease: a sudden rise in temperature; an increase in pulse rate and blood pressure; and aggravation of already present apprehension and tremor or its first appearance.

Eight cases showed extension of the disease a few hours to 5 days after the onset of clinical poliomyelitis. The percentage of complete recoveries in other epidemics ranged from 11–75%; 33 cases (66%) of this series showed paralysis at the onset of the disease. Of the 29 who survived, 11 returned to normal, 6 were slightly involved, 3 moderately so and 9 severely incapacitated—22% of this series.

7. Eight cases showed post-encephalitic symptoms after the acute phase of the illness had passed. They comprised nervousness, irritability and regression in bowel and bladder control. One boy of 8 years tried to commit suicide 8 months afterwards.

OPSOMMING

1. Die sterftesyfer in gevalle van poliomiëlitis wissel van 4–12%, maar kan by wyle so hoog soos 37% wees. In hierdie reeks is 4 pasiënte (8%) oorlede. Bulbêre poliomiëlitis het meer dikwels by die manlike geslag voorgekom.

2. Die tweefasige kliniese patroon het 'n ernstiger prognose as die ander twee patrone gehad.

3. Opvallende encephalitis-simptome, hoë bloeddruk en opmerklike hartversnelling in die afwesigheid van hipoksie het 'n ernstige eindresultaat aangedui.

4. Daar was geen manier om verlamming te antisipeer nie.

5. Die invloed van fisiese bedrywigheid, inentings en inspuitings, tonsillektomie en lang reise word breedvoerig bespreek.

i. Die voortsetting van fisiese bedrywigheid tydens die siekte-aankondigende stadium en die 12 uur wat verlamming voorafgaan, kan aanleiding gee tot 'n hoogs betekenisvolle vermeerdering van ernstige verlamming by volwassenes. In hierdie reeks wat uitsluitend uit kinders bestaan het, is die nadelige effek van fisiese bedrywigheid tydens die siekte-aankondigende stadium ook bevestig.

ii. Die etiologiese rol van binnespierse inspuitings in gevalle van paralitiese poliomiëlitis is nog nie bevredigend vasgestel nie. Die meeste gesaghebbendes is ten gunste daarvan dat geen anti-difterieinspuitings tydens epidemies gegee moet word nie —in sommige streke het witseerkeel-epidemies later uitgebreek. In hierdie reeks was die toevallige verwantskap nie opvallend nie.

iii. Tonsillektomies moet nooit tydens epidemiese poliomiëlitis uitgevoer word nie.

iv. Lang reise is nadelig as poliomiëlitis 'n ernstige omvang aanneem.

6. Sekere kenmerke het 'n uitbreiding van die siekte ingelui: 'n skielike styging van temperatuur; 'n toename in die tempo van die pols en 'n verhoging van die bloeddruk; 'n verergering van die reeds aanwesige vrees en bewing, of die eerste verskyning daarvan.

Ag gevalle het 'n uitbreiding van die siekte getoon binne 'n paar uur tot 5 dae nadat die begin van kliniese poliomiëlitis. Die persentasie pasiënte wat volkome herstel het in ander epidemies het gewissel van 11–75%; 33 gevalle (66%) in hierdie reeks het verlamming by die begin van die siekte getoon. Van die 29 wat die lewe behou het, het 11 na normaal teruggekeer, 6 het geringe verlamming oorgehou, 3 is middelmatig aangetas, en 9 ernstig. Dit kom neer op 22% van die reeks.

7. Ag gevalle het na-encephalitis-simptome geopenbaar nadat die akute fase van die siekte verby was. Dit het bestaan uit senuweeagtigheid, prikkelbaarheid en 'n afname van ingewands- en blaasbeheer. Een seun van 8 jaar het 8 maande later probeer om selfmoord te pleeg.

(To be continued)

ROUNDWORM (ASCARIS LUMBRICOIDES) INFESTATION

ITS SURGICAL PROBLEMS

L. C. J. VAN RENSBURG, M.B., CH.B. (CAPE TOWN)
and
A. E. KARK, B.SC. (RAND), F.R.C.S. (ENG.)
Deparment of Surgery, University of Natal, Durban

Infestation with various worms occurs in over 80% of the African and Indian hospital patients admitted from Durban and surrounding districts and presents a formidable therapeutic problem. A full curative course of treatment with repeated stool examinations in all cases would be prohibitive. Hospital and laboratory services would be strained beyond their capacity and, considering that the living conditions of these people are such that re-infestation is well-nigh certain, such attempts are not warranted.

Ascaris lumbricoides is the commonest helminth found, usually in association with the ova or adult forms of *Trichuris trichiura* and *Enterobius vermicularis*. Hookworm and tapeworm are encountered less frequently.

Usually roundworms cause little observable damage and most patients admitted are found to be incidentally infested. Yet so commonly are they found in association with established surgical syndromes that their role in the aetiology of these conditions will be discussed as well as their possible post-operative complications.

Surprisingly, the important details of the Ascaris life-cycle were not known until 1916, probably because the dangers of hookworm are usually stressed and those of roundworms minimized.[8]

Geographical Distribution. Ascaris lumbricoides is a cosmopolitan parasite found all over the world, but more commonly in the tropics and sub-tropics where the hot and moist atmosphere aids its development.

Morphology and Life Cycle.[8, 16] The Ascaris is whitish-pink in colour with its anterior end thinner than its posterior end, and is one of the largest known human parasites.

The female is 25–35 cm. in length and 5 mm. in breadth. The male is 15–30 cm. long and 2–4 mm. broad and is identified by a ventrally curved posterior end. Three denticulated lips surround the mouth, which leads to a gastro-intestinal tract which is structurally insignificant in comparison with an elaborate genital tract of multiple tubules. In the female this leads to double uteri and in the male to a single ejaculatory duct. In the female Ascaris the uteri open into a single vagina situated at the junction of the anterior and middle of its body. The male has a subterminal cloaca surmounted by two spicules into which the ejaculatory duct leads.

The fertilized adult females lay their eggs in the intestine of man the host, at a rate of up to 200,000 per day.[41] These are passed in the stool and deposited in the soil. Embryonation takes 9–13 days under optimal conditions. The embryonated eggs are very resistant to cold and can survive in 5% formalin for years. Direct sun is harmful to the eggs, but shaded warm and moist soil is ideal for embryonation.

There is no intermediate host and the ova (ingested with contaminated food and especially from the dirty fingers of children) enter the human stomach and pass on to the small bowel where the intestinal juices digest the covering membrane of the egg and the larvae are set free. They burrow their way through the intestinal wall to enter the mesenteric lymphatics and venules and reach the lungs via the liver and right side of the heart. In the lung capillaries, which appear to play an essential role in their development, further growth of the larvae occurs over several days, their length increasing from 200–300 μ to 10 times this length. They penetrate the lung capillaries, enter the alveoli and migrate via bronchioles, bronchi and trachea to the epiglottis where they are swallowed, to reach the stomach where they are resistant to gastric juice and pass on to the intestine, so completing the cycle. At this stage they are 2–3 mm. long, and mature in 2–2½ months to adult worms. Their life span is estimated to be about a year.

This is the normal sequence of events but from the lung capillaries they may pass to the left side of the heart and from there to the general circulation to reach the lymphatic glands, thyroid, thymus, spleen, brain, spinal cord, kidney, placenta and foetus. An Ascaris has been found in the cavity of the human heart.[6]

Pathogenesis. The number of Ascarides in the bowel varies from one to several hundred but usually there are 8 to 10.

The Ascarides feed on the intestinal chyme and, although they bite into the intestinal mucosa, it is generally thought that they do not feed on blood,[16] although some workers disagree with this. Malnutrition is said to occur because there is interference with assimilation of protein in heavy infestation. Japanese workers have found that school children infested with Ascarides were smaller than non-infested children.[6]

The commonest manifestation of these worms is an asymptomatic passage of ova or adult worms in the stools. Adults in an infested population accept this state of affairs and periodically medicate themselves with a variety of home remedies or standard anthelminthics and purgatives.

Anaemia is frequently encountered, usually of a microcytic iron-deficiency type, particularly amongst Indian patients. Here the part played by worms is undisputed. The severely anaemic patients are usually heavily infested with many varieties, hookworm being prominent.

In a number of cases the ova, larval forms or adult worms produce symptoms in the form of allergic reactions, local mechanical blockage or abdominal conditions which are usually serious and may be fatal.

Allergic reactions occur during the invasive stage while the larvae are moving through the lung capillaries, and consist of skin rashes, watering of the eyes and nose, headache and cough.[41] Ascaron, a protein substance contained in the coelomic cavity, is liberated when the worm dies and may give rise to an allergic phenomenon. A specific type of hepatic allergic response, seemingly limited to children between 18 months and 3 years, has been described.[42] It consists of the formation of numerous yellow-white nodules on the liver which histologically show local inflammatory reaction with eosinophilia surrounding the larvae of Ascaris. These authors describe this type of hepatic involvement as an allergic hyperergic response where the liver is the main organ affected.

An Ascaris pneumonia occurs in children who are heavily infested, the larval forms producing haemoptysis and coughing spasms, and secondary infection is common.

There is a variable eosinophilia in all stages of Ascaris infestation, ranging from 5–25%.

The restless adult Ascarides wander widely, wriggling themselves into every orifice of the body. They enter the stomach, oesophagus and nasopharynx. From here they choose either to emerge through the mouth or enter the Eustachian tube and, where there is a perforated eardrum, they will appear in the external ear. They may crawl up the nasolacrimal duct into the lacrimal sac to appear at the inner canthus of the eye.[22]

A more serious complication is laryngeal obstruction causing suffocation. There are reports of Ascaris entering the bronchi via the trachea and giving rise to lung abscesses[18] and they have been found in the heart.[6]

As their normal habitat is the small intestine, not surprisingly the common complications are surgical abdominal emergencies. These often grave complications have focussed much attention on the Ascaris in the last 30 years, and the current literature contains case reports from all over the world.

SURGICAL PROBLEMS

These problems in the abdomen are interesting and varied and can be grouped as follows:

1. MECHANICAL INTESTINAL OBSTRUCTION[4, 9, 24, 41]

This is due to convolutes of roundworms in the small bowel, particularly the lower ileum. More than one segment of bowel may be obstructed and not uncommonly 2 or 3 separate masses are palpable in the abdomen, indicating the accumulated worms in the small bowel. The number of Ascarides causing obstruction varies from a few to many hundreds. The local diet of 'mealie rice', a stodgy porridge-like mixture of ground maize pips, combining with Ascarides in the small bowel, not uncommonly causes symptoms of intestinal obstruction such as seen in Case No. 2.

(*a*) *Intermittent Small Bowel Occlusion.* Children react to this usually in a different manner from adults. Typically there is a history of vomiting worms or passing worms in the stools, and the mother usually admits having given the child a vermifuge a few days before. The child complains of abdominal colic and vomiting. Sometimes there is diarrhoea with blood in the stool. The child looks ill and toxic, with a rapid pulse, a fever of 100°–101°F. and leucocytosis with or without eosinophilia. The abdomen may show slight distension and sometimes a ladder pattern. On palpation one, two or three mobile masses are palpable towards either iliac fossa or near the centre of the abdomen. Within a few hours these may change in position and number, often forming only a single mass.

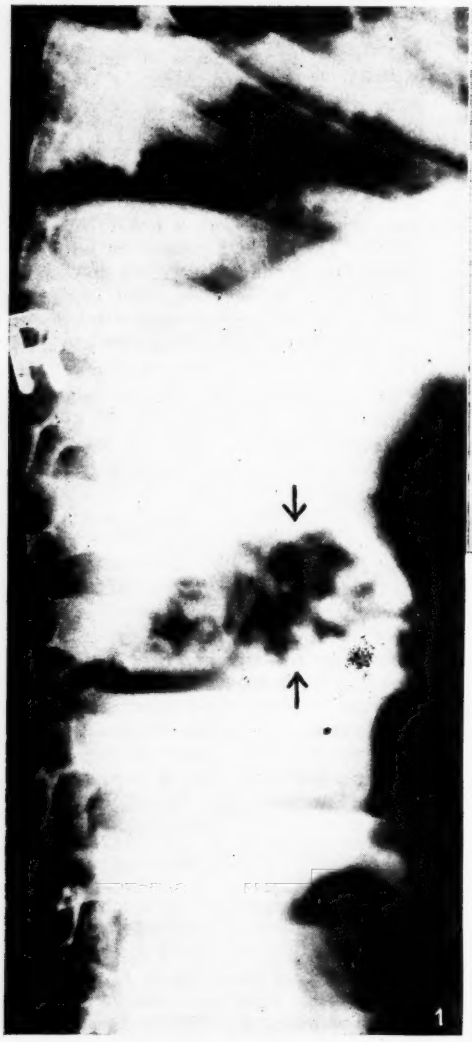
CASE NO. 1

C. M., an African girl aged 8 years, gave a history of abdominal cramps for two weeks which improved when she passed a few roundworms after being treated by her doctor. On admission she complained of severe abdominal cramp, vomiting and absolute constipation for 24 hours.

Her temperature was 99.6° F, pulse 120 per minute, and there were repeated painful colics at 2-minute intervals. Her tongue was moist and clean. The abdomen was not distended and was soft on palpation. A fairly large mass was felt just below the umbilicus with edges difficult to define and tender to the touch. The mass was outlined with ink, and it was found to change its position repeatedly during the next few hours. On rectal examination a mass of worms was felt, but no blood was seen on the examining finger.

Investigation. Blood Hb, 102%; PCV, 42%; MCHC, 36%; White blood cells, 8,000 per c. mm.; Differential Count: Polymorphs, 47%; Lymphocytes, 45%; Monocytes, 5%; Eosinophils, 3%.

A scout X-ray of abdomen showed no radiological evidence of intestinal obstruction or worms. Treatment consisted of pethidine 25 mg. by intramuscular injection on admission, and repeated in 6 hours. The following day pethidine 25 mg. and atropine gr. 1/100 were given. She was given fluids by mouth, starting with 2 fl. oz. hourly for 6 hours then 4 fl. oz. hourly.



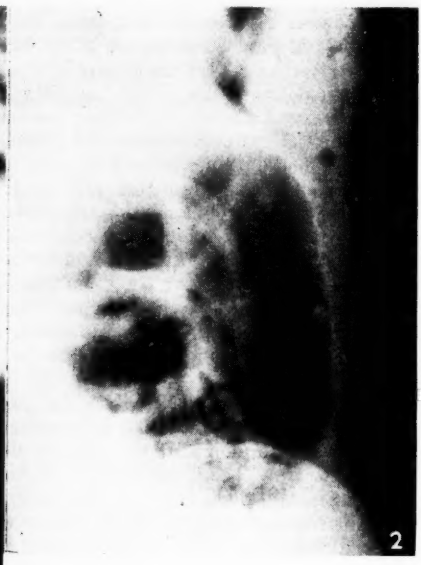


Fig. 1. A lateral scout X-ray showing a mass of worms (arrows) and fluid levels (lower right-hand corner).

Fig. 2. Plain X-ray showing the outline of two Ascarides.

The patient responded well to treatment and 48 hours after admission an anthelminthic was given with a good result on the fourth day, many roundworms being passed *per rectum*. She was discharged from hospital on the eighth day after admission.

In adults there is a difference in symptomatology. Although the history is similar they do not appear ill. The complaint is of vague, colicky, abdominal pains with or without vomiting. There are no toxic symptoms and the temperature is usually normal or slightly raised. The abdomen is usually not distended but one or more typical masses are found which tend to alter in position after some hours. The diagnosis is usually clear but careful observation is necessary.

(*b*) *Complete Intestinal Occlusion.*[4, 14, 27, 35, 37] Worm obstruction is by no means a surgical rarity and deserves a prominent place in the discussion of the aetiology of intestinal obstruction in subtropical countries.

These patients present a typical picture of an acute abdominal emergency with signs and symptoms of intestinal obstruction. Often worms are found in the vomitus and stool. Blood in the stool is said to be a bad prognostic sign.[9] A scout X-ray of the abdomen frequently shows the outline of worms as well as multiple fluid levels (Fig. 1). Barium follow-through studies may demonstrate the worms, as they digest the barium, which may show up their outline and cross striations (Fig. 3).

Post-operatively, in addition to standard treatment, an anthelminthic should be given on the seventh to tenth day.

CASE NO. 2

An Indian male aged 9 years was admitted with a history of slight abdominal pain for 2 weeks. It suddenly became severe the night before admission, when he vomited several times. On examination there was slight fever, the pulse rate was 88 per minute and he had a dry tongue. The abdomen was distended and tender on the right side. A slightly mobile mass was felt in the right iliac fossa, ovoid in shape with fairly well-defined edges. The rectum was empty and there was a little dark blood on the examining finger. No other abnormalities were found. A scout X-ray of the abdomen (Fig. 2) showed clearly outlined worms in the dilated small bowel.

The patient was given pethidine 50 mg. and kept under observation. Three hours later the pulse had risen to 100 per minute, the temperature was 100° F. and the abdomen was a little more distended. The mass was still palpable and was now quite tender. It was decided to operate, as complete intestinal obstruction was now apparent.

Fig. 3. Outline of Ascarides demonstrated by a barium meal.

At laparotomy a segment of ileum about 10 inches from the ileocaecal junction was found to be tightly packed with roundworms. Above this the loops of small bowel were markedly distended. An attempt was made to 'milk' the worms into the caecum, but this was not possible because of the tight impaction and spasm of the small bowel musculature.

Through an ileotomy 60 roundworms with masses of mealie rice were removed. The ileotomy was closed in 2 layers and the abdomen was closed without drainage.

The patient made an uneventful recovery and left hospital on the eighth day to return for anthelminthic treatment.

2. INTUSSUSCEPTION

Girges[18] mentions this as one of the 4 complications ascribed to Ascaris infestation and postulates that the invagination is due to local irritation of the small bowel mucosa with subsequent hyperperistalis. He considers this a rare complication of ascariasis and there are

few references in the literature. Ochsner *et al.*[32] quote McGlannon who described a case of intussusception due to a roundworm in the ileum. Ochsner *et al.* state that one or more worms, not necessarily forming a bolus or blocking the lumen, may start the intussusception. Intussusception in both children and adults is not uncommon in African patients and in most of them no apparent cause is found. The Bantu is so often infested with Ascaris that this is a possible cause, although worms are rarely seen at the site of the intussusception.

Roundworms as a cause of intussusception merit careful consideration and two cases are cited where this form of intestinal obstruction is associated with ascariasis.

CASE NO. 3

An African male aged 11 years was admitted with a history of abdominal pain, diarrhoea and vomiting for 10 days. His mother gave him a 'vermifuge' and he passed a large number of roundworms. On admission he complained of the same pain in the right upper quadrant of the abdomen and of passing watery, blood-stained stools for 3 days.

His temperature was 99° F., pulse 88 per minute, with no signs of dehydration. A fairly well-defined mobile mass could be felt in the right hypochondrium. There was no distension. Rectal examination was normal except for dark faeces on the examining finger.

He was diagnosed as an intermittent worm obstruction, but on the following day he looked ill and dry, with a fever and a rapid pulse. On rectal examination there was dark, foul-smelling, liquid faeces in the rectum and ileocolic intussusception was diagnosed, confirmed at operation. The intussusception reduced easily. No apparent cause was found and the only other positive finding were several Ascarides in the jejunum. Postoperatively the patient progressed satisfactorily and was discharged on the ninth day after operation.

Two possible causes for this intussusception can be postulated.

(*a*) The vermifuge given by the mother before the child's admission could have irritated the Ascarides which in their turn caused hyperperistalsis of the small bowel and subsequent invagination.

(*b*) Increased small bowel peristalsis due to the vermifuge and the purgative given subsequently, which is a customary routine in 'deworming'.

This child suffered from an ileo-colic intussusception associated with Ascaris; but whether or not the Ascaris caused the obstruction, the diagnosis of worm obstruction in such cases is dangerous.

The 3-day history of watery, blood-stained stools in this case is curious, unless the intussusception was a chronic one.

CASE NO. 4

L. R., an Indian boy aged 7 years, was admitted on 26 April 1956, having had a large meal of 'mealie rice' the evening before. He had had nothing since, and at 10 a.m. on the morning of admission had had abdominal colic every 5 minutes; shortly afterwards he started retching. His bowels had not moved for 24 hours. This boy had had numerous attacks of a similar colic in the past and had passed worms.

He was small and pale. A firm, round, tender, smooth mass, constant in position, was felt below and to the right of the umbilicus. Rectal examination was negative. A diagnosis of worm obstruction was made and antispasmodics given. On the following day the mass was easily palpable, but now there was an additional mass above and to the left of the umbilicus. Two days after admission there was still dark-stained blood on rectal examination and the masses were constant. His general condition was satisfactory, with no vomiting and less pain. On the fourth day after admission he vomited twice and was in pain, with visible peristalsis. There were 3 soft, tender masses palpable and the child was given soap water enemas. By the fifth day his clinical condition was deteriorating and, in view of the constant suspicion of an intussusception, a laparotomy was decided upon. A classical ileo-ileal intussusception was found which was gangrenous and irreducible. Resection of 12 inches of ileum and an end-to-end anastomosis were performed. No worm was palpable in any part of the bowel.

He made a slow but uneventful recovery.

3. INTESTINAL OBSTRUCTION DUE TO VOLVULUS[18, 33, 41]

This is a relatively rare complication. Hamilton Bailey mentions it and describes 4 cases. Hamman[19] reviews the cause of volvulus in the small intestine in African patients and mentions Ascaris as an aetiological factor. Dickson,[11] in a review of cases of intestinal obstruction in South African Bantu, found 27% due to volvulus but stated that round-

worms do not cause volvulus. Smirnova[38] reports a case similar to the one to be described. His patient was a girl aged 9 years, diagnosed as an acute intestinal obstruction. At operation a rotation of small intestine through 360° was found which required resection.

The picture is essentially that of a small bowel obstruction with vomiting of worms, Ascarides in the stool, eosinophilia and an abdominal mass as additional features.

The pathogenesis is probably due to a collection of worms lying for some time in a dependent loop of small bowel in which hyperperistalsis occurs and volvulus takes place.[41]

CASE NO. 5

An Indian female aged 22 years was admitted with a history of sudden onset of severe cramp-like pain in the epigastrium for 2 days. The pain came on every half hour. She had been vomiting for 2 days and had not passed a stool and only a little flatus. She was 20 weeks pregnant.

She doubled up with each bout of epigastric pain. The temperature was 102° F. and the pulse 96 per minute. Her abdomen was not distended beyond the limits of a 20 weeks' pregnancy but was tender all over, especially to the right of the umbilicus. There was a positive release pressure sign. The rectum was empty. No other abnormalities were found. A diagnosis of high intestinal obstruction was made and a laparotomy performed.

There was a little free fluid in the peritoneal cavity. A loop of small bowel packed with roundworms was found to have formed a volvulus involving about 14 inches of the jejunum. After untwisting, the bowel was found to be viable and the roundworms were easily milked out of the segment towards the caecum.

She made an uneventful post-operative recovery and was discharged on the eighth post-operative day.

4. ACUTE APPENDICITIS[16, 18, 27]

The appendix is one of the many cavities the Ascaris can enter.

Milwidsky,[27] in an excellent review of the subject, states that although Ascaris-infested patients may develop acute appendicitis unrelated to the worms which may be present in the appendix, or adjoining ileo-caecal region, a true Ascaris appendicitis does occur by supervention of infection in an ulcer produced by the worm.

The signs and symptoms resemble those of appendicitis and even at operation the surgeon may not be aware of an Ascaris in the appendix and be surprised by the pathologist's report of an appendix containing a cross-section of an Ascaris. Usually worms are found in the neighbouring small bowel and a rigid appendix with a tightly impacted Ascaris is diagnostic of the condition.

CASE NO. 6

B. S., an African female aged 25 years, was admitted to the Gynaecology Department of King Edward VIII Hospital on 16 July 1955. A surgical opinion was requested on the same day as it was thought that she had acute appendicitis.

She complained of sudden, severe, burning, epigastric pain which had started the day before and had spread all over the abdomen. There was slight nausea but no vomiting. She had given birth to her second child 3 months earlier and had not menstruated since.

She was distressed and in obvious pain. Her temperature was 101.5° F., pulse 110 per minute. Urticarial wheals were scattered on the abdomen and all over the rest of the body. Abdominal movement was restricted. There was marked tenderness in both iliac fossae, more so on the right side. No masses were palpable but there was generalized rigidity and a positive release pressure sign. On rectal examination there was tenderness on both sides, with soft faeces in the rectum. On vaginal examination the uterus could not be felt. The cervix and both fornices were tender but an acute salpingitis with pelvic peritonitis was considered unlikely.

The urticaria was suspicious of a massive *Ascaris lumbricoides* infestation, but peritonitis secondary to a perforated appendix could not be ruled out. The patient was submitted to laparatomy that same day.

There was no free fluid or pus. The small bowel was congested and contained many roundworms. A roundworm was found tightly impacted in the appendix, which was a little red. The uterus and adxena were normal. The Ascaris was pushed out of the appendix and an appendicectomy performed. The abdomen was closed.

The post-operative course was uneventful and she was given an anthelmenthic before being discharged on the tenth post-operative day.

5. MECKEL'S DIVERTICULITIS[18, 26]

Acute Ascaris Meckel's diverticulitis and perforation of a Meckel's diverticulum with roundworms in the peritoneal cavity have been described. Milroy and Goonawardena[26] state that only 3 cases of perforation of a Meckel's diverticulum with roundworms in the peritoneal cavity have been described and they recorded 2 cases. Smirnova[38] describes a case of a young child who died soon after admission and was found at necropsy to have a large diverticulum (20 cm. long and with the diameter of the ileum) filled with Ascaris and causing an intestinal obstruction. A case of Ascaris Meckel's diverticulitis and perforation is described.

CASE NO. 7

An Indian male aged 5 years was admitted with a history of intermittent cramp-like abdominal pain and absolute constipation for 3 days, and vomiting for 2 days. There was a history of passing roundworms in the stool. In hospital the child vomited several roundworms.

The child looked toxic and dehydrated. His abdomen was distended with ladder pattern formation and visible peristalsis. There was tenderness on the right side but no masses could be felt. The rectum was empty. There were no other significant findings.

The patient was given intravenous fluids and a soap-water enema. The enema yielded many roundworms and faeces. The abdomen was now less distended and less tender. The enema was repeated after one hour and more worms were passed. The child became free of pain and the abdomen was only slightly tender. The following day there was slight distension but no pain or tenderness. There was a rise of temperature and pulse and a few moist crepitations at the left base of the lung. The child was given penicillin 250,000 units 6-hourly. The following day the patient was more ill with a distended abdomen. A scout X-ray of the abdomen showed small bowel obstruction with fluid levels and worms visible in the small bowel (Fig. 1).

Laparotomy was performed in spite of a left basal bronchopneumonia. The small bowel was grossly distended to a point in the terminal ileum where an infected Meckel's diverticulum was found with a stricture of the ileum in this region and a small perforation at the base of the Meckel's diverticulum. The adjacent bowel was inflamed. Several inches of ileum were resected and an end-to-end anastomosis performed. No roundworms were found in the peritoneal cavity and no adult worms were seen near the inflamed area, but several were palpated higher in the bowel. Histology of the specimen showed a Meckel's diverticulum diffusely ulcerated with numerous Ascaris ova in the exudate. Ulceration was also present in the adjacent small intestine.

The patient was extremely ill post-operatively and spent 6 weeks in hospital before recovery and discharge.

CASE NO. 8

The diffuse ulceration of the Meckel's diverticulum and adjacent bowel was probably due to Ascaris. The perforation at the base of the diverticulum occurred in one of these ulcers, and the infection superimposed on the narrowed ileum caused distension of the small bowel and subsequent devitalization of this area.

6. CAECAL DIVERTICULITIS

Milwidsky[27] has described a case of a caecal diverticulum with subacute inflammatory changes. The diverticulum was packed with Ascarides and was resected.

7. PERFORATION OF BOWEL

It is stated[27] that Ascaris can cause perforation of the bowel by damaging the mucosa with its strong denticulated lips, which cause a small ulcer. When infection occurs in this the Ascaris may find its way into the peritoneal cavity. The worms can penetrate pre-existing ulcers such as found in typhoid fever.[40]

M. M., an African male aged 39, was admitted to the McCord Hospital on 1 May 1955 with a sudden severe upper abdominal pain which radiated over the whole abdomen, particularly down the right side. He had not had anything like this before. On examination by the admitting doctor his pulse was 90 per minute, temperature 98.4° F., blood pressure 150/70 mm. Hg. The abdomen showed board-like rigidity with generalized release tenderness and absent bowel sounds. A diagnosis of perforated duodenal ulcer was made.

The abdomen, when seen within a few hours of admission, was slightly distended with a little tenderness in the epigastrium but

no guarding or rigidity; bowel sounds were present. At this time it was felt that the symptoms might have been due to gastritis. On the following day his symptoms returned and he now had board-like rigidity, generalized tenderness and absent bowel sounds, and it was apparent that a viscus had perforated.

At operation there was a large amount of turbid free fluid in the peritoneal cavity and this was sucked out. The stomach and duodenum were normal. On opening the lesser sac to explore the posterior wall of the stomach, Mr. Large was astonished to find a roundworm free in the lesser sac though there was no free fluid present. Systematic exploration of the small bowel showed a small round perforation of the jejunum, 12 inches from the duodeno-jejunal junction. This bore all the hallmarks of an acute duodenal perforation. A few roundworms were felt inside the small bowel. The perforation was sutured and the patient made an uneventful recovery.

Mr. Large's opinion is that when the patient was first admitted, a perforation had occurred, but that by the time he was seen some hours after admission, the roundworm had become lodged in the opening, so enabling him to recover from his symptoms and prevented further leakage. The following day it is presumed that the roundworm worked its way through the opening and that the return of his symptoms was due to the outpouring of more jejunal contents. In some curious way the roundworm must have found its way through the foramen of Winslow, although there might have been other small openings into the lesser sac which were not seen.

A case of perforation of the stomach is quoted by Moore.[28] In addition to perforation of intestinal wall, it is stated that the roundworm can make an oblique track through the wall with its entrance and exit points separated by some inches[27] and the small hole then seals itself off.

8. BILIARY OBSTRUCTION[14, 18, 41]

This complication has received much attention in recent years. The Ascaris may be found in the common bile duct, cystic duct, gall bladder, hepatic ducts and in abscesses in the liver. Fink[14] mentions a case of hepatic infarction due to the presence of a roundworm in the hepatic duct causing pressure on the hepatic artery and subsequent thrombosis. Up to 35 roundworms in the hepatic duct have been found on one occasion. Symptoms of biliary colic may be transient as the worms move on. The severity of the signs and symptoms depend on how long the worms spend in the ducts or how tightly they become impacted. Ova may form the nucleus of calculus formation.

The clinical findings are those of either incomplete or complete intra-biliary obstruction and a confusing variation can occur in the same patient as the worms change position. It is well recognized that the Ascarides convey organisms up the biliary tract, giving cholangitis and hepatic abscesses. Liver function tests show a variable picture of hepatocellular damage and obstructive jaundice.

The serious nature of biliary Ascariasis is illustrated in the following case.

CASE NO. 9

An Indian female aged 25 years was admitted on 18 August 1955 with a history of pain in the epigastrium and vomiting for 5 days. This started soon after giving birth to a child at home. The pain was intermittent and radiated to her back. The vomitus contained some roundworms. She had noticed that her urine was dark and her stool pale in colour.

She was found to be markedly jaundiced. Her abdomen was tender in the right upper quadrant and the liver enlarged. The gall bladder was not palpable. There were no other abnormal findings.

Urine: Albumin, + + +; Bilirubin, a trace.

Stool: ova of Ascaris; Stercobilin + + +.

Liver Function Tests: Direct van den Bergh, +.

Serum bilirubin, 35 mg. per 100 c.c. Alkaline phosphatase, 40 K.A. units. Total protein, 6.1; albumin, 1.3%, globulin 4.8%. A : G ratio, 0.3 : 1.

Cephalin cholesterol 24 hours, 0; 48 hours, + +.

Zinc turbidity 4 units.

Blood urea, 400 mg. per 100 c.c.

These findings were suggestive of biliary obstruction and the raised zinc turbidity and alkaline phosphatase indicated liver damage. On the second day after admission the patient suddenly died.

At post-mortem examination the liver was enlarged and the intra-hepatic bile ducts were obstructed by roundworms. Ascarides were found wedged in the common bile duct but not in the gall bladder, which was grossly

distended. Histology of the liver showed early suppurative cholangitis. In the small intestine several small haemorrhagic ulcers were found.

9. PANCREATITIS[23, 34, 41]

Roundworms in the duodenum may enter the pancreatic duct via the ampulla of Vater and produce the picture of acute pancreatitis. Witenberg[41] lists this as one of the aetiological factors of acute pancreatitis. A case of acute abdominal pain has been reported in a 30-year-old domestic servant in whom a diagnosis of acute appendicitis or gastric perforation was made.[34] At operation there was extensive fat necrosis and a grossly swollen pancreas. She died a day later and at necropsy an Ascaris was found effectively blocking the pancreatic duct and turning round into the duct of Santorini. Novis[31] reported a case with intermittent attacks of abdominal pain resembling biliary colic and at operation the pancreatic duct was slit to find a worm there evidently producing partial obstruction. After removal the patient was quite well. Moore[28] records the autopsy findings in a child where pathologically the body and tail of the pancreas resembled acute pancreatitis. In the pancreatic duct in the body 2 Ascaris worms were found and one in a cyst of the tail. Histologically the changes were compatible with those of acute pancreatitis.

10. ASCARIASIS OF STOMACH AND DUODENUM

Worms may venture into the stomach and apparently live there for some months, impervious to gastric secretion. The varied symptoms of dyspepsia and gastritis are present until relieved by vomiting or the downward passage of the worms. Milwidsky[27] reports 2 cases with severe symptoms and signs suggestive of a perforated ulcer. One patient was saved operation by vomiting worms on the way to theatre. The other patient was operated on and nothing was found apart from worms in the duodenum. Subsequently he had further attacks of pain and marked rigidity which were relieved by enemata and anthelminthics.

Ascarides in the duodenum[12] occur with cramp-like epigastric pain and vomiting and may, if present in large numbers, suggest the picture of pyloric stenosis. X-ray appearances are characteristic with the outlines of the worms visible in the duodenal cap.

11. DEPOSITION OF ASCARIS OVA IN THE PERITONEAL CAVITY

Africa and Garcia[1] recorded a case where embryonated Ascaris eggs were found in the mesenteric tissues of an adult woman. Jenkins[21] removed greyish-white nodules from peritoneal adhesions in a 4-year-old patient with intestinal obstruction and found Ascaris ova surrounded by fibrosis on histological examination. Cooray[10] reviewed an interesting case where embryonated ova of Ascaris were found in the hernial sac of a 3-year-old child.

12. ASCARIS IN THE FALLOPIAN TUBE

Beekhuis[5] found a single Ascaris in the right Fallopian tube of a 44-year-old woman. At operation the ileo-caecal region was found to be adherent to the right adnexa, indicating the site of previous inflammation, and he concluded that the Ascaris reached the Fallopian tube via the appendix.

Stelling and Guay[39] record a case with 5 Ascarides in a Fallopian tube and one in the pouch of Douglas. Murray[29] found a live Ascaris in the Fallopian tube, but does not describe its source.

13. ASCARIASIS OF THE URINARY TRACT

Roundworms have been described in the bladder.[7] The patient complained of acute dysuria for 24 hours. On catheterization, an Ascaris was withdrawn and more worms were withdrawn the following day. No recto-vesical fistula was present.

Worms have been described in the urethra and the kidneys.

CASE NO. 10

G. M., an African male aged 39 was admitted on 19 October 1956, having been stabbed with a knife 15 hours before.

There was a half-inch laceration above the symphysis publis to the right of the mid-line and there was tenderness in the hypogastrium and dullness to percussion. At operation an extraperitoneal tear of the bladder was found and closed in 2 layers and two, 2-inch intraperitoneal bladder tears were closed in 2 layers. The peritoneal cavity was filled with blood-stained urine, but no perforation of bowel was seen. The fluid was removed and a suprapubic cystostomy performed.

During the next week the patient was ill with a high fever and sweating. The abdomen was distended and tender and there were no

bowel sounds. Intravenous fluids and gastric secretions were continued. On the eighth day the suprapubic wound appeared infected, and by the twelfth day a grey, sloughing ragged, wound edge was seen around the suprapubic tube. The wound itself was soft and macerated Three days later a roundworm was seen emerging from the opening in the bladder and the following day 2 further worms were found lying in the sloughing area.

The patient had passed urine normally during the previous 5 days, but had stopped normal micturition during the 24 hours preceding the removal of these 2 worms. Presumably they had blocked the internal meatus. The worms suggested a diagnosis of vesico-intestinal fistula, and close examination revealed that, in fact, the opening on the skin led in 2 directions—one into the bladder and, one, slightly higher up, into the abdominal cavity. A sinogram showed the top opening communicating with the small bowel. Cystoscopy revealed an acutely inflamed bladder, but no fistulous opening.

At the time of operation there appeared to be no bowel damage, but the patient had had urine in his peritoneal cavity for 15 hours. Presumably the bowel became adherent near the bladder and the fistula then formed which opened in the granulation tissue of the suprapubic drainage wound and so allowed the worms to reach the bladder.

DISCUSSION

The asymptomatic passage of ova or worms found incidentally in hospital surgical patients is usually disregarded until the patient is due to leave hospital. Before discharge an anthelminthic drug is given, either piperazine citrate ('Antipar' or 'Helmazine') or 'Crystoids' (hexylresorcinol crystals in gelatin capsules). Hexylresorcinol is efficient and relatively non-toxic; 0.6 g. is given to children and 1 g. to adults, on an empty stomach, the capsules being swallowed and not chewed as the drug irritates the buccal mucosa. After 2 hours a saline purge is given and this routine is effective in 90% of cases.[13] This can be safely repeated in 3 days. The patient is then discharged with instructions to return to Out-Patients for a stool examination.

This treatment usually clears the bowel, but re-infestation on returning to the same home environment is usual. Carbon tetrachloride, when used for hookworm, is irritant to Ascarides and several cases of obstruction and death have been recorded following the use of this drug. However, no anthelminthic is effective in killing larvae during their migration through the body.[40]

Moderate and severe iron deficiency anaemias are frequently seen and after anthelminthic treatment, repeated, if necessary and checked by stool examinations, iron is prescribed by mouth or by intramuscular injections. This is a major problem of public health and education.

The surgical complications due to ascariasis are common in a helminth-infested population. Children presenting with abdominal colic and symptoms of intermittent intestinal occlusion are a difficult problem. The patients show signs of toxicity and usually give a history of having taken a vermifuge with or without a purgative some days before.

The characteristic abdominal finding is one or more mobile, tender, putty-like masses in the iliac fossae or the central abdomen. These should be marked with a pen so that some hours later any relative change in position can be seen. A change in number and position is characteristic of worm impaction. The difficulty arises when the diagnosis is in doubt. It is our experience that a diagnosis of intermittent worm obstruction should not be made unless this characteristic picture is seen and provided a scout X-ray shows no evidence of fluid levels indicating obstruction. Too frequently serious conditions such as intussusception or complete intestinal occlusion are present, which may or may not be due to the worms, and conservative treatment in these circumstances is fraught with danger.

Spastic ileus, a condition of prolonged spasm of a segment of intestine, usually the lower ileum, is quoted by Aird[2] as being due on occasion to a mass of parasites, and has been observed following the migration up and down the gut of a living Ascaris. Experimentally, spasm has been induced by dead Ascarides.[1] The term is not often used as a clinical diagnosis, but there is no doubt that this localized spasm occurs, and Aird quotes an impressive variety of causes.

The clinical picture in children with colic and palpable masses is in many cases probably this condition of spastic ileus, where the bowel goes into spasm due to irritation of mucosa by the Ascaris.[8]

In these cases conservative treatment is indicated, with anti-spasmodics such as pethidine and atropine, varying the dose according to age. A soap and water enema and adequate

oral fluid intake are prescribed and a careful watch maintained for symptoms of complete intestinal obstruction. Anthelminthics should not be given as dead and dying worms will impact in the lower ileum and cause complete obstruction. Romburg[35] has reported success by treating patients in this acute stage with liquid paraffin and anthelminthics daily for 7 days. In his opinion, oiled dead worms are more easily passed. In his series, however, one case obstructed completely.

Once the acute symptoms have subsided, a vermifuge is given after 2–3 days, with the patient still under observaton.

In those cases with complete intestinal obstruction with vomiting, distended abdomen and fluid levels visible on X-rays, worms are commonly found in the vomitus and stool and on X-ray worms are frequently seen outlined in a distended loop of bowel. Immediate exploration is indicated. The worms are found tightly packed in the small bowel and are not necessarily confined to only one segment. Palpation of a single worm gives the feel of a stiff piece of wire lying within the lumen, and is not as soft and pliable as would be expected. The blood supply to the bowel may be interfered with and necrosis may occur, requiring resection of the affected loop.[9, 16] Villalpando, at a Conference under the chairmanship of Gomez[9] mentions 12 such cases occurring in a relatively short period. In one patient, 365 worms were removed and in 5 cases bowel resection was required.[9]

The bowel should not be opened if it can be avoided, as manual removal of worms is akin to pulling on a ball of spaghetti and tends to force the bowel into tonic spasm. The worms should be milked downwards into the large bowel, so loosening the obstruction. If this is not possible, an ileotomy should be performed near the apex of the bolus and the worms removed. More than one ileotomy may be required to complete the removal and the openings closed in the standard 2-layer fashion. Current Russian literature contains numerous reports of obstruction due to Ascaris, and Smirnova[38] is of the opinion that enterotomy should not be performed. He advises immediate resection with end-to-end anastomosis.

When symptoms and signs suggesting obstruction are present, the presence of worms in the stool or vomitus must be regarded as incidental, and not diagnostic of worm obstruction, as has been claimed.[27] The common conditions such as acute appendicitis and intussusception must be considered first and treated as such. The diagnosis of worm obstruction can then be an operative one and dealt with if found.

The role of Ascaris in producing obstruction such as intussusception and volvulus is difficult to demonstrate. Such cases are documented and undoubtedly do occur, but these are so uncommon that the diagnosis of occlusion due to worms should be made with caution. The finding of the worms is incidental at operation, and the primary condition should be dealt with in the standard way.

Case 7 illustrates a diagnosis of worm obstruction where a perforated Meckel's diverticulum was not treated by operation for 2 days in the face of this diagnosis. Case 3 illustrates an intussusception left for 24 hours with a diagnosis of intermittent worm obstruction. Case 4 was carefully observed for 3 days with a typical worm mass in the right iliac fossa which moved its position. Blood on the examining rectal finger was regarded as part of the picture of worm infestation, particularly in view of the typical story of a vermifuge and a meal of 'mealie rice' preceding admission. Laparotomy was finally decided on as the clinical condition was deteriorating, but the diagnosis of worm obstruction was persisted in. At operation a classical gangrenous intussusception required resection, and there was no evidence at operation to show that, in fact, worms were responsible for the condition.

Acute appendicitis and Meckel's diverticulitis with perforation due to roundworms is recognized. In some cases of appendicitis the histological examination shows ova in the mucosa with evidence of thickening and previous attacks of inflammation. Adult worms may become impacted and cause an obstructive appendicitis.

Perforation of the jejunum by Ascaris is reported here, and there are a few similar reports in the literature. Moore[28] records a case of a 13-year-old boy admitted with peritonitis and who died soon after. He was found to have an Ascaris in the peritoneal cavity from a perforation in the stomach. Milwidsky[27] states that Ascaris is capable of penetrating the bowel wall either transversely or obliquely, so that the points of entrance and exit lie several centimetres apart. He quotes Schloessman[36] who reported 6 cases where the worms were found in the peritoneal cavity without any recognizable lesion of the bowel wall; and Freudenthal,[15] who reported one such case. More frequently the penetration of the bowel results in a peritonitis and Ludlow[25]

described a case as well as Gilberti (2 cases) where a perforation was found and worms seen in the peritoneal cavity. These are the only references to this occurrence and it is undoubtedly seldom seen.

Perforation of suture lines by worms following standard operative procedures involving stomach, small and large bowel is usually stated to be a danger,[2, 32, 41] but the recorded cases of such perforation are few. Milwidsky[27] describes a case where 3 fresh ulcers were found in the duodenum near the duodenal stump after gastrectomy. There was one ulcer near the suture line which was intact and the small bowel was packed with Ascarides. He quotes Plew, who described a case of migration of Ascaris through a 4-day-old gastro-enterostomy suture line. Hofmeister described a post-gastrectomy breakdown due to Ascaris, and Milwidsky[27] quotes von Genser who had a fatal case following appendicectomy due to perforation of the suture line. Smirnova[38] also describes this as occurring 5 days after a gastrectomy. Peritonitis developed and, on re-exploration, 11 worms were found in the stomach; one had emerged through the disrupted duodenal stump.

The incidence of stab wounds of the abdomen in this hospital is high and perhaps 80 such cases are treated each year by laparotomy and suture. Yet in these patients, the large majority of whom have worms or who pass ova in the stools, no case has yet been seen of digestion or perforation of the suture line by these worms. It is interesting to speculate that the adult worm might gain exit by the incidental trauma of the stab wound and so account for the finding of embryonated eggs in the peritoneum; but in the cases reported where these ova were found, no such antecedent trauma was known.

Similarly, standard bowel and stomach resections have been followed by disruption of the suture lines no more frequently than elsewhere. In those cases where such a breakdown has occurred, the poor nutritional state of the patients, particularly of those with neoplasms, and technical difficulties or deficiencies, more readily explain the finding of roundworms in the peritoneal cavity or their emergence through a suture line where the worms have found an easy and convenient exit.

Certainly the presence of worms in these African and Indian patients has been no deterrent to any operative procedure and their every-day occurrence is regarded as an incidental finding with little or no post-operative significance.

Biliary tract obstruction has frequently been described, and the typical picture is one of an intermittent obstruction brought about by the alternating impaction of wriggling worms. Surgical removal is necessary. The worms must be removed from the common duct or by milking them down into the duodenum and down the small bowel.

The presence of Ascaris ova, particularly embryonated ova in the peritoneal cavity, is difficult to explain. Embryonated ova are laid only after impregnation of the female Ascaris. Therefore it is more probable that the ova come from the adult female in the bowel itself and that these ova then migrate through small perforations, than that a female Ascaris enters the peritoneal cavity and lays the eggs. The only recorded cases of adult worms being found free in the peritoneal cavity without visible signs of perforation of the bowel are those of Schloessman[36] with 6 cases and Freudenthal[15] with one case. The latter suggests that the adult worm may have developed from a larva in the peritoneal cavity. No such cases appear in the recent literature and no such finding has been encountered here. The other possibility is that larvae reach the left side of the heart and thence the peritoneum and develop in this ectopic situation; but in that case there must be at least one male and one female larva to produce further embryonated ova. Cooray[10] postulates that this strange oviposition occurs by transit through a patent inter-atrial or inter-ventricular septum.

In the many laparotomies done annually in a heavily infested population it would be expected that if larvae do gain access to the peritoneum by way of the systemic circulation, the finding of larvae or adult worms would occasionally be encountered, but this has not been the case. It must be assumed that if larvae do reach the peritoneum by the systemic circulation and produce embryonated ova, then these larvae do not survive in this position. Larvae have been reported causing disturbances in other ectopic situations such as kidney, brain and spinal cord.[40]

SUMMARY

Roundworms are found in over 80% of the non-White population of the Durban area.

The life cycle of *Ascaris lumbricoides* is described.

Most patients found to have ova and worms in the stool are asymptomatic. Eosinophilia is commonly present. Moderate or severe microcytic anaemia is common. Anthelminthic

treatment using hexylresorcinol is safe and effective.

Intermittent intestinal occlusion with colicky pains and fever is commonly seen in children who frequently give a history of having taken a vermifuge some days before. The diagnosis must be made with caution and careful observation is necessary. These patients should be treated conservatively with antispasmodics. No anthelminthics should be given until the severe symptoms have subsided, as dead and dying worms tend to produce complete occlusion.

Acute intestinal occlusion is seen in both adults and children, but the diagnosis must only be made when a putty-like, mobile mass is palpable in association with worms visible on X-ray.

The vomiting of worms or their passage in the stool is to be regarded as an incidental finding and not pathognomonic of worm obstruction.

The diagnosis of intestinal occlusion due to worms is a dangerous one when dealing with acute perforated viscera or intussusception.

Volvulus due to roundworms is recorded and a case is reported, but this is rare.

Intussusception due to roundworms is the possible explanation in some cases, the mass of worms acting as an apex; but few authenticated cases are reported and none is described with the apex consisting of a bolus of worms. Intussusception is a more commonly seen cause of obstruction in African and Indian children up to the age of 12 years than in European children, but the relationship to worms is not proven. Nevertheless, worms may act as an irritant causing hyperperistalsis and so initiate the process. Two cases are described where the findings were associated in one case with Ascarides in another level of the bowel, and in the other with vomiting of worms.

Perforation of bowel by Ascarides is recorded, but case reports are few and this complication is rare. A case is described (and 4 others have been recorded at this Hospital) of a perforation of the jejunum.*

Roundworms have been described in every body cavity and duct including those of the central nervous system. Biliary obstruction tends to be intermittent with fluctuating jaundice and variable results of liver function tests. The worms may cause pancreatitis and Meckel's diverticulitis. Acute appendicitis may result from impaction of an Ascaris and a case is reported.

The presence of the worms in the genito-urinary system is due either to direct entry via the female genital tract, so reaching the Fallopian tubes, via the urethra to the bladder or via a fistulous connection with the bowel.

The alleged propensity of the Ascaris to eat its way through catgut suture lines is not borne out in our experience. This complication has not been encountered in a heavily infested population where the incidence of open and closed visceral perforation due to trauma is high. A few cases reported are better explained on the basis of the worms escaping after the perforation has occurred. Thus the presence of worms at operation presents little operative or post-operative danger in this respect other than the recognized complications of intestinal occlusion.

The finding of embryonated ova in the peritoneal cavity is difficult to explain. No cases of larvae or adult worms lying free in the peritoneal cavity at operation have been seen at this hospital, unless the bowel has been perforated; and only 2 authors in the literature have reported such an occurrence. Whether ova, larvae or adult worms can penetrate the bowel wall through a minute hole without symptoms is not known.

OPSOMMING

Rondewurms word aangetref by meer as 80% van die nie-blankes in die Durbanse gebied.

Die lewenskringloop van *Ascaris lumbricoides* word beskryf.

Die meeste pasiënte in wie se ontlasting die eiers en wurms aangetref is, is asimptomaties. Eosinofilie is gewoonlik aanwesig. Matige of ernstige mikrositiese bloedarmoede is 'n gewone verskynsel. Wurmafdrywende behandeling waarby daar gebruik gemaak word van heksielresorsinol is veilig en doeltreffend.

Intermitterende ingewandsokklusie met koliekagtige pyn en koors word dikwels aangetref by kinders, en uit hul geskiedenis blyk gewoonlik dat hulle 'n paar dae tevore 'n wurmafdrywer geneem het. Die diagnose moet met sorg gedoen word, en sorgvuldige waarneming is nodig. Hierdie pasiënte moet konserwatief met krampweringsmiddels behandel word. Geen wurmafdrywer moet toegedien word voordat die ernstige simptome bedaar het nie, want die dooie en sterwende wurms het 'n neiging om algehele okklusie te veroorsaak.

*Since the submission of this MS., 4 similar cases have been recorded in this Hospital. A tiny, clean, punched out hole in the bowel has been found—3 times in the jejunum and once in the ileum with minimal inflammatory reaction for ¼ inch round the perforation. In 2 cases a living adult Ascaris was found free in the peritoneal cavity, but in the other 2 cases no apparent cause for the perforation was found other than the presence of worms elsewhere in the bowel. Microscopy showed a non-specific inflammatory reaction of the edge of the perforation without evidence of Ascaris ova.

Akute ingewandsokklusie word aangetref by sowel volwassenes as kinders, maar die diagnose moet alleen gedoen word wanneer die stopverfagtige, mobiele massa voelbaar word in assosiasie met wurms wat met behulp van X-strale sigbaar is.

Die opbring van wurms of hul aanwesigheid in die ontlasting moet beskou word as 'n bykomstige bevinding en nie as patognomonies van wurmobstruksie nie.

Die diagnose van ingewandsokklusie te wyte aan wurms is gevaarlik wanneer die dokter te doen het met akute geperforeerde ingewande of derminstulping.

Dermknope wat deur rondewurms veroorsaak is, is reeds aangetref, maar dit is 'n seldsame verskynsel.

Derminstulping wat aan rondewurms te wyte is, is in sommige gevalle 'n moontlike verduideliking, en die massa wurms kan as die toppunt optree; maar min bevestigde gevalle is gerapporteer, en geeneen met 'n toppunt bestaande uit 'n klein ronde massa wurms is tot dusver beskryf nie. Derminstulping is 'n meer gewone oorsaak van obstruksie by naturelle- en Indiër-kinders tot die ouderdom van 12 jaar as by blanke kinders, maar die verwantskap met wurms is nog geensins 'n uitgemaakte saak nie. Nietemin is dit moontlik dat die wurms as 'n prikkelmiddel optree wat hiperperistalsis veroorsaak en die proses aan die gang sit. Twee gevalle word beskryf waar die bevindings in die een geval geassosieer was met Ascarides in 'n ander peil van die derm, en in die ander geval met die opbring van wurms.

Perforasie van die derm deur Ascarides is reeds teëgekom, maar die verslae is min en hierdie verwikkeling is 'n seldsame verskynsel. 'n Geval waar die middeldeel van die dunderm geperforeer was, word beskryf. Vier dergelike gevalle is by hierdie besondere hospitaal waargeneem.

Rondewurms is reeds aangetref in iedere liggaamsholte en buis, insluitende dié van die sentrale senuweestelsel. Galobstruksie het 'n neiging om intermitterend te wees met wisselende geelsug en veranderlike resultate as lewertoetse gedoen word. Die wurms kan alvleesklierontsteking en die divertikulitis van Meckel veroorsaak. Akute blindedermontsteking kan die gevolg van 'n Ascaris-beklemming wees, en so 'n geval is reeds gerapporteer.

Die aanwesigheid van die wurms in die geslags-urinêre stelsel is te wyte òf aan regstreekse indringing deur die vroulike geslagskanaal, ten gevolge waarvan die wurms die Fallopiaanse buise bereik en vandaar deur die urinebuise na die blaas gaan, of deur 'n fistelkonneksie na die ingewande.

Die beweerde neiging van die Ascaris om sy weg deur dermsnaarhegtingslyne te vreet, word nie deur ons ondervinding gestaaf nie. Hierdie komplikasie is nie aangetref by 'n groep mense wat ernstig besmet is nie, en by wie die voorkoms van oop en geslote ingewandsperforasie wat aan trauma toegeskryf kan word, hoog is. Die paar gerapporteerde gevalle word makliker verduidelik op die grondslag dat die wurms ontsnap het nadat die perforasie reeds plaasgevind het. Die aanwesigheid van wurms tydens 'n operasie bring dus min operasie- of na-operasie-gevaar mee, met uitsondering van die erkende komplikasies van ingewandsokklusie.

Die ontdekking van embrionale eiers in die peritoniumholte is moeilik om te verduidelik. Geen gevalle van larwes of volgroeide wurms wat los in die peritoniumholte lê, is tydens 'n operasie by hierdie hospitaal aangetref nie tensy die derm vroeër geperforeer was; slegs 2 skrywers in die mediese literatuur het so 'n voorval gerapporteer. Of eiers, larwes of volgroeide wurms die derm deur 'n klein gaatjie kan binnedring sonder simptome is nie bekend nie.

We should like to thank Dr. S. Disler, the Medical Superintendent of King Edward VIII Hospital, for permission to report these cases; and Mr. P. Large, M.S., F.R.C.S., who kindly made the details of Case 8 available. We also thank Mr. C. J. Lockett for the X-ray photographs and Mrs. J. Terry-Lloyd for secretarial work.

REFERENCES

1. Africa, C. M. and Garcia, E. Y. (1936): J. Phillipp. Med. Assoc., **16**, 461.
2. Aird, I. (1949): *Companion in Surgical Studies*, 1st ed. Livingstone & Co. Ltd.
3. Bailey, H. (1952): *Emergency Surgery*, 6th ed., pp. 432–433. John Wright.
4. Barber, F. (1947): Brit. Med. J., **1**, 49.
5. Beekhuis, J. J. (1948): Nederl. T. Geneesk., **3**, 2756.
6. Boetigger, C. and Werne, J. (1929): J. Amer. Med. Assoc., **93**, 32.
7. Carsten, M. (1927): Dtsch. Med. Wschr., **53**, 411.
8. Chandler, A. C. (1955): *Introduction to Parasitology*, 9th ed., pp. 440–447. John Wiley.
9. Gomez, F. and Torre, J. A. (1954): J. Pediat., **45**, 478.
10. Cooray, G. H. (1951): Ann. Trop. Med. Parasit., **45**, 62.
11. Dickson, P. R. (1950): S. Afr. Med. J., **24**, 705.
12. Druckmann, A. and Schorr, S. (1945): Acta. Med. Orient. (Tel-Aviv), **4**, 385.
13. Faust, E. C., Sawitz, W., Tobie, J., Odom, V., Peres, C. and Lincicombe, D. R. (1939): J. Parasitol., **25**, 241.
14. Fink, H. (1948): Amer. J. Trop. Med., **28**, 585.
15. Freudenthal (1923): Med. Klin., **11**, 19. (*Quoted by* Milwidsky, H.)
16. Gradwohl, R. B. H. and Kouri, P. (1948): *Clinical Laboratory Methods and Diagnosis*, 4th ed., pp. 278–300. Vol. 3, *Parasitology and Tropical Medicine*. Mosby.
17. Gilberti (1920): Policlin. Roma, **51**, 27. (*Quoted by* Milwidsky, H.)
18. Girges, R. (1934): J. Trop. Med. Hyg., **37**, 296.
19. Hamman, J. (1951): S. Afr. Med. J., **25**, 241.
20. Hofmeister (1913): Zentrbl. f. Chir., **30**, 1179. (*Quoted by* Milwidsky, H.)
21. Jenkins, M. Q. and Beach, M. W. (1954): Pediat., **13**, 419.
22. Kaplan, C. S., Freedman, L. and Elsdon-Dew, R. (1956): S. Afr. Med. J., **30**, 791.
23. Kortzeborn, A. (1924): Munch. Med. Wchnschr., **71**, 473.
24. Landsborough, D. (1946): Brit. Med. J., **2**, 461.
25. Ludlow (1927): Chin. Med. J., **41**, 134. (*Quoted by* Milwidsky, H.)
26. Milroy, P. and Goonawardena, D. F. (1953): Amer. J. Surg., **85**, 243, 370.

27. Milwidsky, H. (1945): Acta. Med. Orient. (Tel-Aviv), **4**, 363.
28. Moore, McK. P., Jnr. (1954): South. Med. J., **47**, 9.
29. Murray, H. E. (1926): J. Obstet. Gynaecol. (Brit. Emp., **23**, 448.
30. McGlannon, A. (1916): South. Med. J., **9**, 977. (*Quoted by* Ochsner, de Bakey and Dixon[32]).
31. Novis, Lt.-Col. (1923): Brit. J. Surg., **10**, 421.
32. Ochsner, A., De Bakey, E. G. and Dixon, J. L. (1949): Amer. J. Dis. Child., **77**, 389.
33. Patoir, G. and Le Doux, J. (1942): Presse. Med., **50**, 643.
34. Rigby, H. M. (1923): Brit. J. Surg., **10**, 419.
35. Romburg, R. G. (1953): E. Afr. Med. J., **30**, 251.
36. Schloessman (1914): Beitr. Z. Klin. Chir., **90**, 531. (*Quoted by* Milwidsky, H.)
37. Skapinker, S. (1949): Brit. J. Surg., **37**, 110.
38. Smirnova, K. A. (1952): Khirurgiya, **4**, 53.
39. Stelling, R. and Guay, A. J. L. (1936): J. Amer. Med. Assoc., **107**, 2046.
40. Strong, R. P. (1945): *In* Stitt's *Diagnosis, Prevention and Treatment of Tropical Diseases*, 7th ed. Blakiston.
41. Witenberg, G. (1945): Acta. Med. Orient. (Tel-Aviv), **4**, 363.
42. Behrer, W. R. and Klingberg, W. G. (1951): J. Pediat., **38**, 635.

NOTES AND NEWS : BERIGTE

Drs. Samuel, Komins, Denny, de Villiers and Berezowski (Radiologists), wish to inform their colleagues that they have opened X-ray consulting rooms for out-patients on the Third Floor, Clarendon Centre, 4 Park Lane, Johannesburg.

Drs. Samuel, Komins, Denny, de Villiers en Berezowski (Radioloë), wens hulle kollegas in kennis te stel dat hulle spreekkamers vir X-straal ondersoek, vir buite pasiënte, op die derde verdieping te Clarendon Centre, Parklaan 4, Johannesburg, geopen het.

Mr. Cyril D. Kisner, M.B., Ch.B., Ch.M. (Rand), urologist, has moved from 103 Lister Buildings, to Suite 500-501 Medical Arts Building, Jeppe Street, Johannesburg. (Telephones: *Rooms*: 23-8200; *Residence*: 43-5300).

Mnr. Cyril D. Kisner, M.B., Ch.B., Ch.M. (Rand), uroloog, het die adres van sy spreekkamers verander na Medical Arts Building 500-501, Jeppestraat, Johannesburg. (Telefoon nommers: *Kamers*: 23-8200; *Woning*: 43-5300).

PREPARATE EN TOESTELLE

SORBACEL

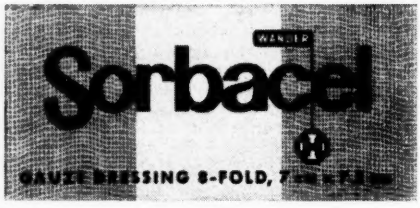
NUWE ABSORBEERBARE HEMOSTATIESE GAAS

Westdene Products (Pty.) Ltd. kondig aan dat voorrade *Sorbacel*, die nuwe absorbeerbare hemostatiese gaas, geproduseer deur Wanders, van Switserland, tans beskikbaar is.

Om bloeding stop te sit wat nie chirurgies gekontroleer kan word nie, is 3 soorte absorbeerbare stof in die afgelope 10 jaar ontwikkel—fibrienspons geweek in trombien, gelatienspons en oksisellulose. Eersgenoemde twee stowwe word betreklik stadig geabsorbeer en bring die produksie van korrelweefsel mee. Derhalwe kan hulle alleen vir aseptiese operasies gebruik word. Oksisellulose, daarenteen, word veel vinniger deur die weefsels geabsorbeer sonder enige merkbare selreaksie. In die vorm waarin dit tot dusver beskikbaar gestel is, kon oksisellulose egter aanleiding tot weefselprikkeling gee weens die suur aard daarvan. Temeer, dit inaktiveer trombien en suur-gevoelige antibiotica soos penisillien, en dit word nie deur etterende weefsel geabsorbeer nie.

Sorbacel is 'n verdere ontwikkeling van oksisellulose in 'n produk wat nie langer die nadele van die oorspronklike suur-materiaal besit nie. Dit word verkry deur die oksidasie van katoengaas, en die latere neutralisering met kalsium van die poliuroonsuur wat gevorm word. Die kalsiuminhoud van die preparaat is ongeveer 6%.

Sorbacel is gereed vir gebruik, en baie maklik en eenvoudig om te hanteer. Dit kan alleen gebruik word, of saam met trombien, en dit word nie klewerig of slymerig selfs wanneer dit deurweek is nie. Die hemostatiese effek daarvan is beter as dié van gelatienspons en van suur-oksisellulose weens die aanwesigheid van kalsium. In normale omstandighede word dit binne enkele dae geabsorbeer. *Sorbacel* veroorsaak geen weefselprikkeling nie, en, anders as suur-sellulose, bevorder dit wondgenesing. Dit kleef intiem aan die wond vas, en, indien nodig, kan dit saam met enige antibiotiese oplossing aangewend word.



Sorbacel wat blou van kleur is om dit van gewone gaas te onderskei, is verkrygbaar in kompresse, 7 sm. x 7.5 sm., en in stroke, 3 sm. x 1 meter. Dit word steriel en gereed vir gebruik verskaf, en moet nie opnuut gesteriliseer word nie, want die gaas is gevoellig vir hitte. *Sorbacel* moet in 'n koel plek bewaar word.

Nadere inligting in verband met *Sorbacel* is verkrygbaar van die alleenverspreiders vir Suid-Afrika, Westdene Products (Pty.) Ltd., Essanby-gebou, Jeppestraat, Johannesburg.

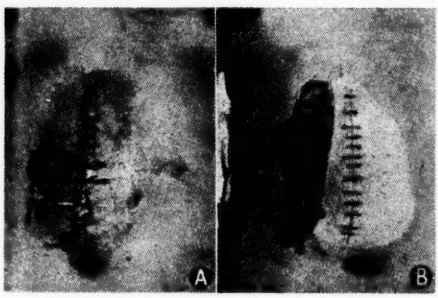
LIQUIDO PLAST

DIE NUWE VERSTUIFBARE VERBINDSEL, VELBESKERMER EN GENEESMIDDELDRAER

Liquido Plast word met behulp van 'n verstuiwer aangewend, of, so nie, word dit aangesmeer; dit word dan vinnig droog en vorm 'n plastiese laag. Die aanwending daarvan is buitengewoon maklik en vinnig, en geen spesiale vernuf is nodig daarvoor nie. Lug kan die *Liquido-Plast*-laag deurdring, maar bakterieë kan dit nie doen nie. Vrye en onbelemmerde asemhalings- en gewrigsbewegings word deur die treffende buigbaarheid van hierdie laag moontlik gemaak.

Die stowwe wat in *Liquido Plast* vervat is, is fisiologies onskadelik, met die gevolg dat geen algemene simptome van siekte of plaaslike prikkeling—insluitende dié wat aan gevoeligheid te wyte is—geproduseer word nie. Hierdie stowwe is poliviniel-derivate, opgelos in asetoonetielasetaat.

As Verbindsel: As verbindsel bied die steriele, plastiese *Liquido Plast*-laag doeltreffende beskerming teen meganiese besering en sekondêre infeksies. Die deurskynende laag kleef alleen aan die vel in die onmiddellike nabyheid van die wond vas. Dit oorbrug die wond self, met die gevolg dat granulasie nie belemmer word nie. Die wond kan te alle tye waargeneem word sonder dat dit nodig is om die laag te verwyder.



A: *Liquido Plast*-laag 13 dae na maagreseksie.
B: Afskilfering van laag en deursnyding van velhegtings.

Aanwendingsvelde: (*a*) Ná hemostase as 'n eerste verbindsel vir alle operasiewonde, bv. appendektomie, herniotomie, laparotomie, torakotomie, maagreseksie, nefrektomie, skildklieruitsnyding, plastiese chirurgie, kopwonde, boudnaathegting, ens.

(*b*) As tweede verbindsel vir wonde waar daar kontra-indikasies vir die gebruik van *Liquido Plast* as eerste verbindsel is (bv. in gevalle waar 'n infeksie met anerobe kieme vermoed word).

(*c*) Vir daardie dele van die liggaam waar die aanlê van ander verbindselsoorte moeilikheid oplewer.

(*d*) As bevestigingsmiddel vir verbindsels.

(*e*) Vir die bestuiwing van die operasieplek voordat 'n operasie uitgevoer word. Dit bespaar steriele draperinge, en die snit kan regstreeks deur die plastiese laag gemaak word. Die operasie kan begin 30 sekondes nadat die laag aangewend is.

(*f*) Vir die verbinding van brandwonde.

Beskerming van die Vel: Danksy die onoplosbaarheid van die *Liquido Plast*-laag in water en die goeie huidverdraagsaamheid daarvan selfs by allergiese persone, skyn dit asof hierdie laag besonder geskik is vir die bedekking van dele van die vel wat aan gevaar blootgestel is, sowel as vir die bedekking van besoedelende velmiddels.

Aanwendingsvelde: (*a*) Vir die beskerming van die vel teen die prikkeling wat deur afskeidings veroorsaak word, bv. in gevalle van dreinering, ulcus cruris, en fistels. (Moet dit egter nie aanwend nadat die prikkeling reeds sy verskyning gemaak het nie).

(*b*) Vir die beskerming van velsiektes wat gevoelig vir water is.

(*c*) Vir die bedekking van besoedelende velmiddels (gensiaanviolet, briljante groen, *Cignolin*, teer, ens.).

As Geneesmiddeldraer: Die byvoeging van aktiewe stowwe by die *Liquido Plast*-laag het geneesmiddels met 'n langdurige effek opgelewer, want die medisyne word in sulke omstandighede stadig vrygestel. Die gebruik van *Liquido Plast*-laag as 'n geneesmiddeldraer maak dit ook moontlik om buitepasiënte te behandel sonder om hul onderklere te besoedel. Geen ekstra verbindsel is nodig nie.

Aanwendingsmetodes: *Liquido Plast*-T (met die byvoeging van 10% koolteer vir die behandeling van alle velsiektes, ekseem, neurodermatitis en psoriase wat op teer reageer).

Liquido Plast-P (met die byvoeging van 0.5% *Cignolin* (Bayer-Leverkusen) vir die behandeling van alle soorte psoriase wat op *Cignolin* reageer).

Let Wel: Die laag word na 'n paar dae opgelig deur normale afskilfering van die vel, en kan dan maklik verwyder word. Die laag kan ook te eniger tyd met asetoon verwyder word. Hegtings kan dwarsdeur die plastiese verbindsel verwyder word. Chirurgiese handskoene word nie deur *Liquido Plast* geaffekteer nie.

Alleenagente vir Suid-Afrika: Protea Pharmaceuticals Ltd., Posbus 7793, Johannesburg.

PREPARATIONS AND APPLIANCES

SORBACEL

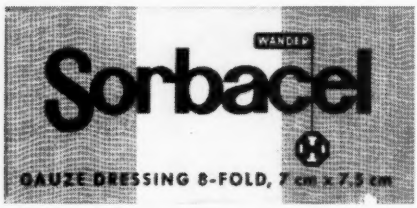
NEW ABSORBABLE HAEMOSTATIC GAUZE

Westdene Products (Pty.) Ltd. announce that supplies are now available of *Sorbacel*, the new absorbable haemostatic gauze produced by Wanders of Switzerland.

To check bleeding which cannot be controlled by surgical means, 3 kinds of absorbable material have been developed in the last 10 years—fibrin sponge soaked in thrombin, gelatin sponge and oxycellulose. The first two substances are absorbed relatively slowly with the production of granulation tissue and can therefore be used only in aseptic operations. Oxycellulose, on the other hand, is absorbed much more rapidly by the tissues and without any appreciable cell reaction. In its hitherto existing form, however, oxycellulose could also lead to tissue irritation due to its acid character. Furthermore, it inactivates thrombin and acid-sensitive antibiotics such as penicillin, and is not absorbed in suppurating tissues.

Sorbacel represents a further development of oxycellulose to a product which no longer exhibits the disadvantages of the original acid material. It is obtained by the oxidation of cotton gauze and the subsequent neutralization with calcium of the polyuronic acids formed. The calcium content of the preparation amounts to about 6%.

Sorbacel is ready to use and easy and simple to handle. It may be applied on its own or with thrombin and does not get sticky or slimy even when saturated. Its haemostatic effect is superior to that of gelatin sponge and of acid oxycellulose, due to the presence of calcium. It is normally absorbed in a matter of a few days. Sorbacel does not cause tissue irritation and, unlike acid oxycellulose, promotes wound healing. It adheres intimately to the wound and, where required, may be applied with any antibiotic solution.



Sorbacel, which is blue in colour to distinguish it from ordinary gauze, is available in compresses 7 cm. x 7.5 cm. and strips 3 cm. x 1 metre. It is supplied sterile ready for use and may not be re-sterilized because the gauze is sensitive to heat. *Sorbacel* should be stored in a cool place.

Further information regarding *Sorbacel* may be obtained from the sole South African distributors, Westdene Products (Pty.) Ltd., Essanby House, Jeppe Street, Johannesburg.

LIQUIDO PLAST

THE NEW SPRAYABLE DRESSING, SKIN PROTECTION AND DRUG CARRIER

Liquido Plast is sprayed on by means of an atomizer, or else spread on, and forms a plastic film after drying rapidly. Its application is extremely simple and quick and requires no special skill. *Liquido Plast* films are air-permeable, but are impassable to bacteria. Their great flexibility permits free and unobstructed respiratory and articular movement.

The substances contained in *Liquido Plast* are physiologically harmless, so that no general symptoms of disease or local irritation—including those due to sensitization—are produced. These substances are polyvinyl derivatives dissolved in acetone-ethyl acetate.

Dressing: The sterile plastic film *Liquido Plast* offers adequate protection as a dressing against mechanical injury and secondary infections. The transparent film adheres only to the surrounds of the wound, and bridges the wound itself, so that granulation is not impeded. The wound can be observed at all times without removing the film.

Fields of Application: (*a*) After haemostasis as a first dressing for all operation wounds, e.g. appendectomy, herniotomy, laparotomy, thoracotomy, gastric resections, nephrectomy, thyroidectomy, plastic surgery, head wounds, perineal sutures, etc.

(*b*) As a second dressing for wounds where the use of *Liquido Plast* is contra-indicated as a first dressing (e.g. in cases where an infection with anaerobic germs is suspected).

(*c*) For those parts of the body where the application of other kinds of dressing causes difficulty.

(*d*) As a fixing agent for dressings.

(*e*) To spray the operating site pre-operatively, thus saving sterile drapes. Incisions can be made directly through the plastic film. The operation can be performed 30 seconds after application of the film.

(*f*) To dress burns.

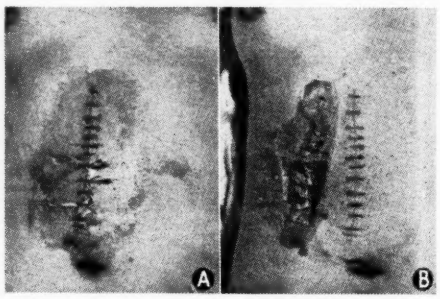
Skin Protection: Thanks to its insolubility in water and good cutaneous tolerance even by allergic persons, *Liquido Plast* film appears to be particularly suitable to cover up endangered areas of skin and soiling skin remedies.

Fields of Application: (*a*) Protecting the skin against irritation by discharged excretions, e.g. in cases of drainage, ulcus cruris, and fistulae. (Do not apply when the irritation has already manifested itself).

(*b*) Protecting dermatoses sensitive to water.

(*c*) Covering up soiling skin-remedies (gentian violet, brilliant green, cignolin, tar, etc.).

Drug Carrier: The addition of active substances to the *Liquido Plast* film created remedies which achieve a protracted therapeutic effect by a steady emission of the medicament. The use of *Liquido Plast* films as drug-carriers also permits of out-patient treatment without soiling underwear. No additional dressing is required.



A: *Liquido Plast* film 13 days after gastric resection.

B: Peeling off of film and cutting of apposition sutures.

Modes of Application: *Liquido Plast*-T (with an addition of 10% of coal tar for the treatment of all dermatoses, eczemas, neurodermatitis, and psoriasis, responding to tar).

Liquido Plast-P (with an addition of 0.5% of Cignolin (Bayer-Leverkusen) for the treatment of all types of psoriasis responding to cignolin).

Note: The film is lifted after a few days by the normal desquamation of the skin and can be removed easily. The film can be removed at any time with acetone. Sutures can be removed through the plastic dressing. Surgical gloves are not affected by *Liquido Plast*.

Sole South African Agents: Protea Pharmaceuticals Ltd., P.O. Box 7793, Johannesburg.